This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

CHRONIC TRAUMATIC OSTEOMYELITIS

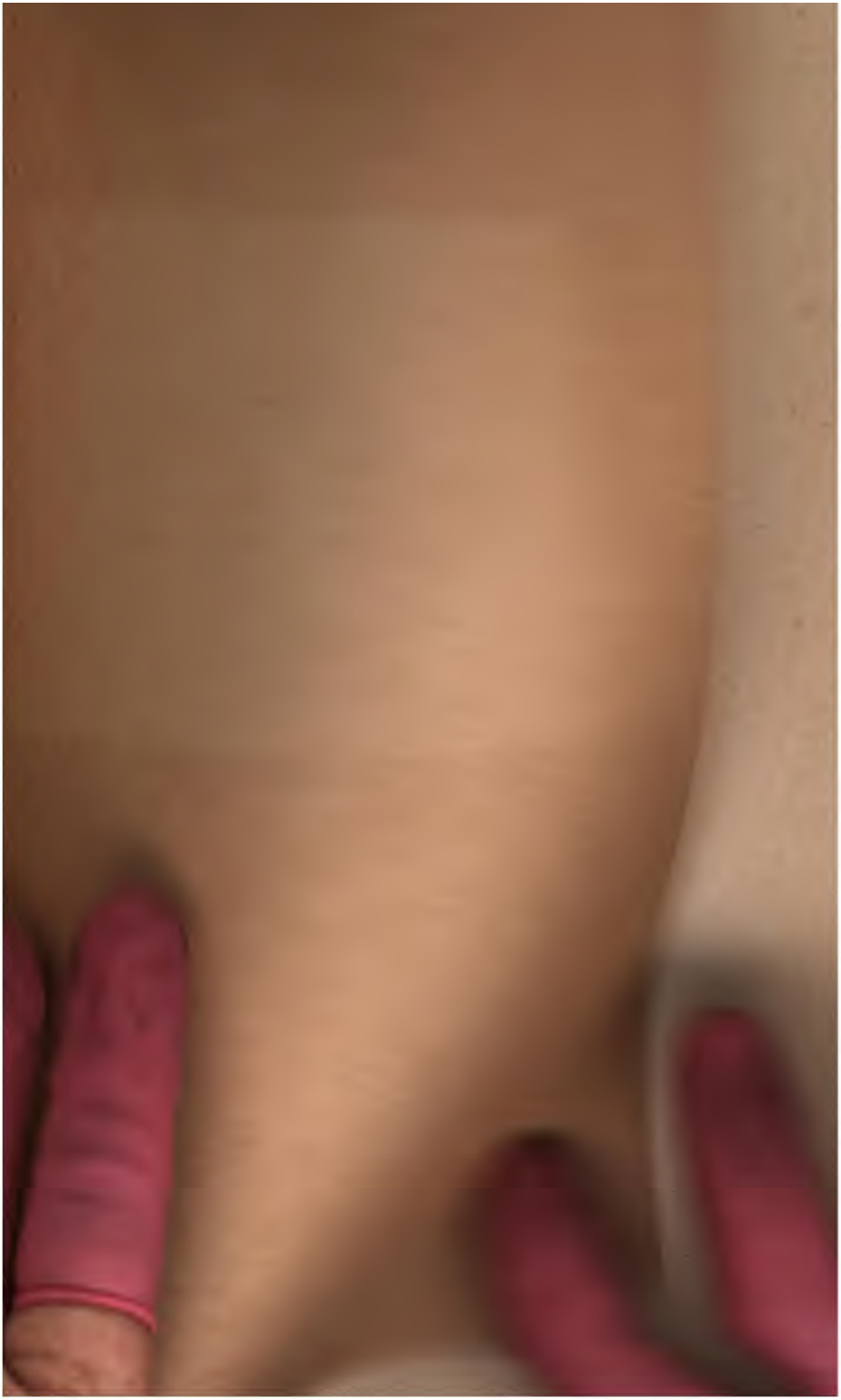
J. RENFREW WHITE

LANE MEDICAL LIBRARY STANFORD

LANE

MEDICAL LIBRARY

LEVI COOPER LANE FUND



CHRONIC TRAUMATIC OSTEOMYELITIS

PLATE VIII.—Chronic osteomyelitis of the femoral shaft ; a large primary sequestrum surrounded by several secondary sequestra lying encased within a sequestral cavity whose walls are of an enormous thickness.

[*Frontispiece*

CHRONIC TRAUMATIC OSTEOMYELITIS

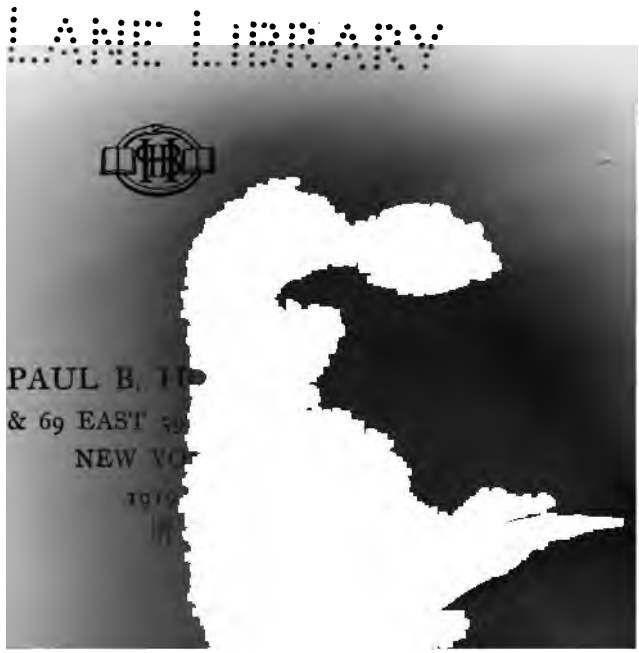
ITS PATHOLOGY AND TREATMENT

BY

J. RENFREW WHITE, M.B. (N.Z.), F.R.C.S.

FORMERLY RESIDENT SURGICAL OFFICER, ROY. NATIONAL ORTHOPÆDIC HOSPITAL, LONDON
FORMERLY SURGEON, TOOTING MILITARY ORTHOPÆDIC HOSPITAL
ORTHOPÆDIC SURGEON, NEW ZEALAND FORCES

WITH TWENTY-FOUR PLATES AND THIRTEEN DIAGRAMS


LANE LIBRARY
PAUL B.
67 & 69 EAST
NEW

[*Printed in England.*]

LANE LIBRARY

CONTENTS

PAGE

INTRODUCTION xi

CHAPTER I

THE PHYSIOLOGY OF BONE 1

CHAPTER II

GENERAL CONSIDERATION OF THE PHENOMENA OF OSTEOMYELITIS 8

CHAPTER III

THE PATHOLOGY OF COMPOUND FRACTURES AND OF CHRONIC TRAUMATIC OSTEOMYELITIS 47

CHAPTER IV

THE EXAMINATION OF CASES OF CHRONIC TRAUMATIC OSTEOMYELITIS 82

CHAPTER V

THE CLINICAL COURSE OF CHRONIC TRAUMATIC OSTEOMYELITIS 87

CHAPTER VI

GENERAL PRINCIPLES OF THE TREATMENT OF CHRONIC TRAUMATIC OSTEOMYELITIS . . . . . PAGE 91

CHAPTER VII

DETAILS OF TREATMENT OF CHRONIC TRAUMATIC OSTEOMYELITIS: DETAILS OF CASES 105

INDEX 136

W58
1919

LIST OF ILLUSTRATIONS

PAGE

DIAGRAM 1.—The three zones of the fracture cavity in a simple fracture with neither displacement nor comminution . . 36

DIAGRAM 2.—The fracture cavity of a simple fracture without comminution, but considerable lateral displacement . . 38

DIAGRAM 3.—The fracture cavity of a simple fracture without comminution but with overlapping of the fragments . . . 38

DIAGRAM 4.—The three zones of the fracture cavity of a simple fracture with comminution but with little or no displacement of fragments 39

DIAGRAM 5.—The fracture cavity of a simple fracture without comminution but with marked displacement and associated with periosteal laceration 39

DIAGRAM 6.—The sequestral cavity of an infected transverse fracture without comminution or displacement . . . 54

PLATE I. CASE 1.—Chronic osteomyelitis of the shaft of the fibula with an antero-posterior tunnel containing several very small secondary sequestra . . . . . *facing* 54

PLATE II. CASE 1.—Showing the result of the radical operation; removal of the sequestra with subperiosteal resection of the outer wall of the tunnel . . . . . *facing* 54

PLATE III. CASE 2.—Chronic osteomyelitis of the shaft of the fibula with a large antero-posterior tunnel. The sequestra have all been previously removed without healing resulting *facing* 54

PLATE IV. CASE 3.—Chronic osteomyelitis of the shaft of the humerus, showing a large cavity lying between anterior and posterior walls . . . . . *facing* 54

PLATE V. CASE 4.—Chronic osteomyelitis of the shaft of the femur; formation of a tunnel by deposit of new bone around a drainage tube . . . . . *facing* 56

PLATE VI. CASE 6.—Chronic osteomyelitis of the shaft of the femur; there has been little comminution but great displacement of the fragments; no formation of a real sequestral cavity; a few secondary sequestra and many infected pieces of metal embedded in all parts of the callus . . . *facing* 56

50316

PAGE

DIAGRAM 7.—The sequestral cavity of a transverse fracture without comminution but with displacement 57

PLATE VII. CASE 7.—Chronic osteomyelitis of the shaft of the fibula; an antero-posterior tunnel containing a small sequestrum . . . . . *facing* 58

PLATE VIII.—Chronic osteomyelitis of the femoral shaft; a large primary sequestrum surrounded by several secondary sequestra lying encased within a sequestral cavity whose walls are of an enormous thickness . . . . . *Frontispiece*

PLATE IX.—Chronic osteomyelitis of the shaft of the humerus; a large tunnel with very thick walls . . . . *facing* 58

PLATE X. CASE 8.—The end result of a case of chronic osteomyelitis of the shaft of the femur; numerous sequestra were removed; sub-periosteal resection of the outer wall of an enormous antero-posterior tunnel; so much bone was removed that fracture of the shaft occurred and rendered possible correction of the deformity. This plate was taken after healing and sound union . . . . . *facing* 58

PLATE XI.—Chronic osteomyelitis of the shaft of the femur; an oblique tunnel with several sequestra . . . . *facing* 58

DIAGRAM 8.—The sequestral cavity of a transverse fracture with comminution but without displacement 59

DIAGRAM 9.—The sequestral cavity of a transverse fracture with comminution and displacement 59

DIAGRAM 10.—The sequestral cavity of an oblique fracture without comminution or displacement 60

DIAGRAM 11.—The sequestral cavity of an oblique fracture without comminution but with displacement 60

DIAGRAM 12.—The open sequestral cavity of an infected fracture with complete comminution of a segment of the bone . . 62

DIAGRAM 13.—Failure of union with absence of sequestral cavity 62

PLATE XII. CASE 9.—Before operation. Cavity formation in the shaft of the tibia . . . . . *facing* 126

PLATE XIII. CASE 9.—After operation. The cavity has been converted into a groove and obliterated by subperiosteal resection of its antero-external wall . . . . *facing* 126

PLATE XIV. CASE 10.—A piece of shrapnel embedded in cancellous tissue of the lower third of the femoral shaft . *facing* 126

PLATE XV. CASE 10.—Shows the cavity that remained after the removal of the foreign body . . . . . *facing* 126

PAGE

PLATE XVI. CASE 10.—After the obliteration of the cavity by subperiosteal removal of its antero-internal wall, healing occurred. Plate taken after healing . . . . . *facing* 126

PLATE XVII. CASE 10.—A cavity in the left Os Calcis after the removal of a piece of metal . . . . . *facing* 128

PLATE XVIII. CASE 10.—A cavity in the right Os Calcis after the removal of a piece of metal . . . . . *facing* 128

PLATE XIX. CASE 10.—Subperiosteal removal of the inferior wall of the cavity has resulted in its obliteration and healing; considerable subperiosteal formation of new bone . . *facing* 128

PLATE XX. CASE 10.—Subperiosteal removal of the superior wall of the cavity has resulted in its obliteration and healing; considerable subperiosteal formation of new bone . *facing* 128

PLATE XXI. CASE 11.—Antero-posterior view of the upper end of the tibia completely excavated by the process of caries *facing* 130

PLATE XXII. CASE 11.—Lateral view before operation *facing* 130

PLATE XXIII. CASE 11.—After operation; obliteration of the cavity by removal of its anterior wall followed by plastic operation . . . . . *facing* 130

PLATE XXIV. CASE 11.—Lateral view after operation of the case shown in Plate XXII. . . . . *facing* 130

INTRODUCTION

"Not enough thought is given to the life which those suffering from osteomyelitis are fated to lead in the future, with continued pain, sinuses constantly reappearing necessitating repeated operations which they must undergo without ever knowing how far away is the promised cure, or whether amputation will not eventually be necessary. Surely it is worth while to take any trouble that will save this long martyrdom."

LERICHE.

SINCE the commencement of the present war, the medical profession, and through reports in the lay press often exaggerated and erroneous, even the general public, have heard much of the success claimed for various operative methods and for numerous antiseptic agents in the immediate disinfection of wounds, or at least in such prevention of development of the inevitably existent infection as allows of their early, and even in some cases, immediate closure. These types of operation and various chemical agents have thus in turn singly or in combination been proclaimed as the solution of the problem, the more or less certain means of realisation of this ideal of disinfection.

These early operations, involving the complete excision of bruised and damaged skin and muscle, the extraction of all septic foreign bodies and extraneous débris, the ensuring of complete hæmostasis, the removal of a variable amount of the comminuted bone, and the institution of adequate drainage, have all one common aim, *viz.*—the reduction to a minimum of all encouragement to the development of the micro-organisms already present in the wound.

These chemical agents, carbolic acid, hypochlorous acid, sodium chloride, magnesium sulphate, soap solution, iodoform, brilliant green, flavine, etc.; various in their nature and

mode of application, acting directly or indirectly, have had one common purpose, *viz.*—the destruction of such germs as remain in the wound after the early operation.

By such different means, but with this one aim of early wound disinfection, certain workers claim to be so far successful in the case of compound fractures, as to have solved the problem of the prophylaxis of osteomyelitis. They claim to be so far successful in the accomplishment of the immediate or rapid disinfection of the wound and seat of fracture, thus converting compound into what, though still open, are practically simple fractures; as to enable the surgeon in some cases to proceed with safety and success to an early and aseptic plating of the fractured shaft, in almost all to avoid the numerous and dangerous sequelæ and the painful and obstinate disabilities of chronic bone disease. Such results are claimed by Leriche to be achieved by a combination of early adequate excision of damaged soft parts with immediate total subperiosteal esquillectomy in fractures of the shaft, with immediate subperiosteal resection of the joint in articular fractures. Relying on Dakin's fluid so used as to remain in intimate contact with every part of the wound and fracture, Carrel puts forth a claim to similar results.

In England, however, we have as yet seen no results in fracture cases comparable to those of Leriche, no results comparable to those of Carrel. Shell and bomb fractures almost without exception have arrived in England cases of acute or chronic osteomyelitis; of the results of prophylaxis of this terrible disease little has so far been seen. Almost the only fractures that arrive "simple" are those that have been "simple," uninfected from the beginning—fractures, the result of rifle and machine-gun bullets and occasionally of shrapnel balls.

That such prophylaxis is possible has again been proved by the statistics of the result of primary suture in compound fractures recently given by Sir Anthony Bowlby,[1] and now

[1] Lecture delivered before the Royal Society of Medicine, London February 13th, 1918.

at the time of writing just published[1]; but these figures refer, as doubtless also do those of Leriche and Carrel, to cases submitted to operation under specially favourable circumstances, cases received early during a quiet time from a quiet battle area, operated on at leisure and with a painstaking thoroughness hardly possible in a time of rush and congestion, and subsequently kept under special observation for a considerable time. Such conditions would seem to constitute a *sine qua non* of the success that can be obtained by such early and radical intervention; but despite these remarkable results it would appear probable that at least the majority of shell and bomb fractures occurring in big battles or in a war of movement will still be destined to remain compound, to develop into cases of chronic osteomyelitis.

However this may be, whatever the future may hold in store, whatever developments in the direction of prevention of bone infection may arise, and on whatever scale they may become practicable, there exist to-day not only in England, but in every belligerent country in the world, tens of thousands suffering the discomforts and dangers inseparable from the constantly or intermittently discharging sinus, the result of a gunshot fracture.

The prophylaxis of osteomyelitis as such, moreover, cannot directly concern orthopædic surgeons in England; but from a humanitarian, a scientific, and also an economic point of view, the adequate treatment leading to early arrest and final cure of this pathological process has become a subject of national, indeed of international importance.

The seriousness and the desperate chronicity of this disease have always been recognised. No chronic non-malignant disease of the extremities keeps in such constant jeopardy the lives and limbs of its victims; its obstinacy will soon become a by-word even among the laity; in very many cases wounds, no sooner healed, break down on the least or without any apparent provocation. Patients discharged from hospital make their reappearance, often within a few days

[1] *Brit. Med. Journal*, March 23rd, 1918, p. 333.

with their old sinus again open; in other cases, although the disease has for months been quiescent and to all appearance finally cured, the return to civilian life and active employment is sooner or later interrupted by a recrudescence of inflammation, or a reopening of a healed sinus; and readmission to hospital with further operation becomes in acute cases urgent, in all ultimately inevitable. The impossibility of spontaneous cure and the failure of such surgical attempts at cure as are inadequate, largely because based on merely vague or inaccurate pathological conceptions, are the cause of the retention and congestion in our military hospitals of thousands of fracture cases in which the bone has been long since firmly united.

Chronic osteomyelitis is not only an evil and a disability in itself, but is fraught with consequences most adverse as regards the treatment and cure of concomitant disabilities. The persistent focus of sepsis and inflammation ever threatening to break out with fresh violence, not only postpones active use of the limb, but even stays the hand of the masseur, thus depriving muscles and joints of the advantages of passive movement and massage. Its chronicity is responsible for the long, sometimes indefinite and often detrimental postponement of such orthopædic operations as nerve suture, corrective osteotomy, resection of ankylosed joints, bone suture, and grafting of ununited fractures and pseudarthroses. Even when healing has been laboriously accomplished, the surgeon is by no means free to proceed at once to deal with the residual disability. The fear of a recrudescence of the infection renders further postponement of any operation absolutely necessary. The occurrence of such post-operative recrudescence, by no means unknown even after the lapse of an apparently safe and prolonged period of waiting—and never so severe as when it takes the form of a return of osteomyelitis—constitutes for the surgeon a source of great anxiety, for the patient a calamity, always serious and often dangerous, loss of limb and even of life being the not infrequent consequences.

Existing then, as it does to-day in Europe, on an already

absolutely unprecedented scale, it would hardly be possible to exaggerate the urgency of the solution of the problem—the early and final cure of chronic osteomyelitis. In the face of these facts the writer makes no apology for the production of this book, the subject matter of which is the pathology and treatment of this disease.

Whether what seems to the writer should constitute the ideal towards the realisation of which treatment should be directed be capable of attainment in all cases is yet to be seen; nearness of approach to it in most cases, however, will depend not only on surgical skill and ingenuity, but more especially on a perfect realisation of the significance of the exact pathological conditions present in each particular case. That many of the methods and operations still in use are doomed to failure from the outset seems to the writer to be due primarily to a failure on the part of the surgeons practising them of a full comprehension of the problems presented by chronic osteomyelitis.

That finality of cure will be obtained in every case in which this ideal seems to have been realised would be as yet premature to claim. That this ideal, however, and the methods of its attainment set forth in this book are based on sound surgical and pathological principles, and that they represent the best available means of cure at our present disposal the writer feels confident.

CHRONIC TRAUMATIC OSTEOMYELITIS

ITS PATHOLOGY AND TREATMENT

CHAPTER I

THE PHYSIOLOGY OF BONE

The General Structure of Bones

Apart from their vessels and nerves and a certain limited amount of ordinary connective tissue, bones are formed of several different and important varieties of tissue :

(1) Bone tissue proper.
(2) Marrow tissue.
(3) A peripheral fibrous vascular membrane, the periosteum.

Bone Tissue Proper.—Like all connective tissues, bone tissue proper is made up of two elements :

(*a*) The vital essential bone cells.
(*b*) The non-vital ground substance or matrix.

The cells of normal, fully developed bone tissue are of one variety ; ovoid, nucleated, with numerous branches or processes, they lie in closely fitting and specially lined spaces of the matrix called **lacunæ** ; the filiform processes of the cells occupy corresponding ramifications of the spaces known as **canaliculi**. There is nothing of special interest or

importance in the chemical constitution of these bone cells.

The matrix consists of an organic basis impregnated or combined with certain mineral salts or bone-earths. These are readily dissolved out and separated from the organic element by the action of dilute hydrochloric acid; such decalcification respects, however, the shape of the bone; a framework remains consisting largely of a protein called ossein, a mucoid and an albuminoid. On the other hand, calcining of bone destroys its organic matter and leaves but a framework of bone-earths that also retain the original shape of the complete bone. The chemical composition of this ash in human bones is given by Carnot[1] as follows:

	%
Calcium phosphate	87·45
Magnesium phosphate	1·57
Calcium fluoride	·35
Calcium chloride	·23
Calcium carbonate	10·18
Iron oxide	·1

Whilst different bones and parts of the same bone yield on analysis a variable proportion of organic and inorganic matter, bone tissue proper is supposed by Hoppe-Seyler[2] on very likely grounds to have a constant chemical constitution of about 75% of inorganic material; the variations in the amount of inorganic matter found on analysing different bones—ranging from about 48% to 70% by weight—are merely the result of differences in the relative amounts of bone tissue proper and of the other tissues present in bone. The writer has found the low figure of 45% of inorganic salts in a portion of the os calcis, whilst in a piece of the tibial crest the figure was 69%.

The matrix, although when taken in the mass probably of a constant chemical composition, is not, however, homogeneous. It is of a lamellated structure due to an arrangement of fine fibres that run through its calcified

[1] Carnot, *Compt. Rend.*, 156.
[2] Hoppe-Seyler, *Physiol. Chem.*, 102-104.

ground substance. The layer of the matrix immediately surrounding the lacunæ and canaliculi is said to have a chemical composition rather different to that of the rest and to be more resistant to the action of hydrochloric acid.

Marrow Tissue exists in all bones, but in varying proportions to the amount of bone tissue proper ; frequently this proportion between the two tissues varies greatly in different parts of the same bone. Marrow is of two chief varieties—in certain bones yellow, due to a great admixture with adipose tissue, in others, where associated with but little fat, its extreme vascularity becomes evident by its red colour. Either form is a richly cellular tissue containing, besides fat cells and the ordinary cells of the blood, three special varieties of cells of its own :

(1) Myelocytes—the essential and most characteristic cells of the marrow tissue. These are granular and amœboid, and are classified according to the staining reactions of their granules as neutrophil, eosinophil, or basophil.

(2) Nucleated erythroblasts, the fore-runners of the erythrocytes of the blood. Like them they contain hæmoglobin ; they are classified according to their size as normoblasts, microblasts, and megaloblasts.

(3) Large giant cells called myeloplaxes ; polynuclear or else with one large ringed nucleus. These are in appearance and probably in fact identical with the osteoclastic cells that are so constantly associated with any process, normal or pathological, of bone absorption.

Marrow tissue is richly vascular and contains numerous thin-walled capillaries and veins ; in every bone by far the greater amount of the actual bone tissue obtains its blood-supply through and from these vessels of the marrow.

The Periosteum is the peripheral or limiting membrane whose functions and powers have been the subject of an historical controversy which has not even as yet finally

subsided. It is a richly vascular fibrous membrane possessing two well-recognised layers the inner of which contains "elastic fibres and blood-vessels in a minute state of subdivision." Besides these layers Macewen describes another of loose areolar tissue lying between the surface of the bone proper and the inner vascular layer of the periosteum. This areolar tissue he describes as constituting a potential space of the greatest practical importance. After full growth of the bone the periosteum becomes very firmly adherent to the surface of the bone, whilst in childhood the connection, except at the epiphyseal lines, is much less intimate. Hence this potential space of Macewen's is of greater significance in childhood than in later life.

According to which of two different modes of arrangement and of two correspondingly different relative proportions of bone and marrow tissues obtain, two different varieties of bone structure are to be recognised—**compact** and **cancellous** bone. Not only in **compact** bone is the amount of marrow tissue reduced to a minimum so that it is far denser and stronger though less vascular than cancellous bone, but it is characterised by a special system of structural arrangement of the bone tissue proper which is perforated by minute Haversian canals about 1/500ths of an inch in diameter. These run parallel to the long axis of the bone, but connect across at frequent intervals by transverse and oblique passages. Round each canal there is a concentric arrangement of the lamellated bone-tissue so that an Haversian system—the unit of compact bone—is formed. The spaces which exist between the contiguous cylindrical Haversian systems are filled up with non-lamellated bone tissue. Each Haversian canal, with its lateral branches contains capillaries, nerve filaments, and lymphatic spaces, the last connecting directly through minute passages with the canaliculi and the lacunæ; each canal contains a little ordinary connective tissue, whilst the larger contain in addition a small amount of marrow tissue.

That part of the compact bone, however, that lies immediately underneath the periosteum has a rather different

structure. It receives its blood-supply from vessels of the periosteum that enter the bone through fine canals called Volkmann's canals. Here there is no Haversian system; hence a difference in the arrangement of the lamellæ which, running parallel to the outer surface of the bone, become the **circumferential lamellæ.** In addition, bundles of white and elastic fibres pass from the periosteum transversely through this peripheral layer—**the perforating fibres of Sharpey.**

Cancellous bone, on the other hand, consists simply of very thin layers of bone tissue proper so arranged as to form a reticular framework, the interstices of which are filled with the highly vascular marrow tissue.

Variations in the architecture of different bones are due to differences in combination of compact and cancellous bone. From this point of view three varieties of bones are to be distinguished:

(1) The **short bones,** such as those of the carpus and the tarsus, and the **epiphyses,** such as the articular end of the long bones or the great trochanter of the femur. In these there is a very thin outer layer of compact bone enclosing a mass of cancellous bone.

(2) The **flat bones,** *e.g.* the ribs, the innominate bones, and the bones of the skull; in these a layer of cancellous tissue lies between two thick plates of compact bone.

(3) The **shafts** or **diaphyses of the long bones** which consist of a thick cylindrical sheath of compact bone tunnelled by a wide canal filled with pure marrow tissue only.

The Development and Growth of Bone

Bone is developmentally ossified connective tissue; certain bones—"cartilage bones"—result from a process of ossification of pre-existing cartilage; others—"membrane bones"—from direct ossification of a form of embryonic connective tissue.

Endochondral Ossification.—There are three recognised stages in this process. The first consists of calcification of the cartilaginous matrix with a definite arrangement of the cartilage cells in rows. Simultaneously there takes place a subperiosteal deposit of lamellæ which, at first fibrous, become calcified and come to include between them certain cells that have been instrumental in their production—osteoblasts. The first real production of bone-tissue is thus subperiosteal. The second stage—the stage of irruption—consists of the ingrowth of the cells of this subperiosteal bone through the calcified matrix of the cartilage which becomes absorbed to form cavities called **medullary spaces.** This absorption process is carried out by large giant-cells or osteoclasts of doubtful origin. In the third stage of development further subperiosteal deposit of bony lamellæ takes place together with similar deposit of new bone on the walls of the medullary spaces. This new bone tissue is not at first characterised by the presence of Haversian systems, whilst in long bones the regular central medullary canal does not form from fusion of the medullary spaces until after birth.

At varying periods, but mainly after birth, separate centres of ossification appear in the epiphyses, and growth of bone takes place both in shaft and in the epiphyses with consequent formation of line or layer of the still unossified epiphyseal cartilaginous plate.

Growth in length of bone takes place in continuous expansion lengthways of these epiphyseal cartilages, increase in the width of which, however, is prevented by progressive corresponding conversion of the newly formed cartilage into bone. After a certain age this growth of the epiphyseal cartilage comes to an end with the result, its own invasion from both sides by bone cells and subsequent conversion into bone. Increase in width of a bone is effected according to some observers by further subperiosteal deposit, according to Macewen by interstitial growth.

Direct or Intramembranous Ossification. — Calcification takes place in a primitive connective tissue that contains

numerous fibres in its ground substance. The cells of this connective tissue become the osteoblasts.

One's whole view of the growth and development of bone, however, and also of the production of new bone in pathological conditions, depends entirely on the rôle that one is prepared to grant to the periosteum. This, again, resolves itself into the question of the origin of the osteoblasts. These have been held to arise from the blood-vessels of the periosteum—in which case one can quite rightly claim osteogenetic powers for that membrane alike under normal conditions of growth and development and under the abnormal circumstances of inflammation and infection. Similar conclusions must logically result from the adoption of the view that the osteoblasts are converted or modified connective tissue cells of the periosteum. Such, indeed, has been the general view and teaching of physiologists, and, since the classical work and writings of Ollier, of most surgeons and pathologists. Against such a view, however, must be set the opinion and experiments of Sir William Macewen [1] who regards all osteoblasts in diaphyseal ossification as the result of proliferation of the cartilage cells, in pathological osteogenesis, as derived from the ordinary cells of bone tissue proper. Hence he denies absolutely to the periosteum any osteogenetic powers; all normal growth and development of bone and all reparative or defensive or adaptive formation of new bone tissue in inflammation result according to Macewen from the proliferation of the interstitial cells of cartilage and bone respectively.

[1] Macewen, *The Growth of Bone*, 1912.

CHAPTER II

GENERAL CONSIDERATION OF THE PHENOMENA OF OSTEOMYELITIS

INFLAMMATION being the local reaction of tissue to an irritant, it follows that the pathological phenomena present in an inflamed infected area at any one moment represent the local net result at that moment of the struggle between the tissues and the infection. Such result might be summed up in terms of gain and loss on the part either of the tissues or of the organisms; or, again, as a balance struck between the destructive and the defensive or reparative elements of the situation. In the case of most tissues these two summaries would amount to one and the same thing, the positive defensive phenomena being due solely to the effort of the tissues and the destructive purely and directly the result of the bacterial activity. Such cannot be said, however, to be true of inflammatory disease of bone. Because of peculiarities of structure and nutrition, bone tissue, when inflamed, often becomes the seat of destructive changes that are the result in part at least of the tissue reaction, and that cannot wholly be attributed in all cases to the direct destructive action of the infection. In all cases of inflammation in soft parts where death does not ensue nor surgery intervene, the destructive processes, sooner or later coming to an end, are wholly replaced by reparative, and the period of healing commences; although in some cases of very chronic infections neither the destructive nor the reparative process is capable of making headway against the other, destruction and healing progressing over long periods of time simultaneously and on much the same scale. In such cases surgery

can often come to the aid of the defence and succeed in turning the balance against the infection. In many cases of acute or subacute infection, however, repair goes on to the completion of healing and cure; in others a point is ultimately reached beyond which further repair becomes for various reasons impossible, a condition only too common in cases of bone disease.

The conditions arising, then, in inflammatory diseases of bone are in many important respects not only different from but also more complex than those obtaining in other more vital tissues. Not only does Nature, in her defensive efforts, seem to a certain extent to play into the hands of the enemy, but her attempts at healing, to a greater extent perhaps than in other tissues, remain halting and ineffective. In most cases of inflammatory disease of bone the result of infection, complete and final healing demands the co-operation of a surgeon armed with an absolutely accurate realisation of the actual pathological conditions present in each case, and a knowledge of the requirements and means whereby alone the elements obstructive to healing can be removed and the reparative utilised and assisted.

The bases from which arise these peculiarities of the inflammatory phenomena in bone tissue are two, one anatomical, the other physiological; the former is to be found in the structural arrangement of bone by which the essential vital protoplasmic element with its blood-vessels is enclosed within the walls of a rigid non-vital framework of mineral salts; the latter, physiological, in the special activities and powers possessed by this vital element in the two opposite directions of gradual absorption of the rigid element surrounding it and of its increase by further deposit of mineral salts.

The former structural arrangement is responsible for the extreme liability to massive necrosis of acutely inflamed bone tissue; the latter physiological properties give rise to two pathological processes, one, osteoporosis or caries, destructive, producing rarefaction of bone; the other, osteosclerosis, defensive and reparative, resulting not only

by an increase of mineral salts in a greater relative density of structure, but in many cases in an actual formation of new bone or osteogenesis.

These three processes, necrosis, caries and osteosclerosis, represent the essential basis of difference between the phenomena of osteomyelitis and those of inflammation elsewhere in the body. Apart from their occurrence the phenomena of tissue reaction, retrograde and regenerative alike, are identical in nature if not in degree with those by inflammation in other tissues; vaso-constriction followed by a vaso-dilatation affecting arteries, veins, and capillaries; an acceleration giving way to a slowing and finally to complete stasis of the blood-flow; the phenomena of exudation of concentrated lymph; diapedesis and the escape of red corpuscles; thrombosis of vessels leading to areas of necrosis,—all these normal inflammatory phenomena are present; phagocytosis, bacteriolysis, and suppuration occur; apart from osteosclerosis, even the reparative phenomena to a certain extent and in many cases are not different. Moreover, these three peculiarities of osteomyelitis are strictly analogous to corresponding inflammatory processes in soft parts, and represent a difference that from the pathological, though not from the therapeutic standpoint, is to a certain extent more apparent than real.

We must now proceed to study in detail each of these three phenomena and especially their significance in the pathology and treatment of compound fractures and traumatic osteomyelitis.

The Destructive Phenomena of Osteomyelitis—Necrosis and Caries

These two processes are respectively analogous to the destructive inflammatory phenomena of gangrene and ulceration in soft tissues.

Necrosis.—Where an appreciable amount of bone dies *en masse* it is said to undergo necrosis. Necrosis is therefore gangrene, or sloughing of bone tissue; the term applies to

the death of any macroscopic quantity of bone. In necrosis only the vital bone elements die; the inert matrix, rigid from impregnation or combination with bone-earths, undergoes no immediate change. All changes that take place in or around such dead bone tissue take place subsequently and gradually and really form no essential part of the phenomenon of necrosis.

The Causes of Necrosis.—Necrosis of bone owns the same causes as gangrene of soft parts. Hence it may arise: from direct devitalisation by trauma, by chemical or by bacterial action; indirectly from interference with the nutrition of the bone-cells; most commonly from a combination of these two factors.

Necrosis from direct devitalisation by trauma, uncomplicated by bacterial action, is but rarely seen. The early aseptic stage of deep burns with direct bone involvement and death furnishes about the only example where necrosis from trauma is met with clinically. How often it occurs and to what extent, if at all, in simple fractures cannot be determined.

Necrosis from the action of chemicals occupies a similar indeterminate position; it is never seen except where complicated by sepsis, and in such cases it is impossible to tell how far the necrosis is due to the former and how far to the latter agent. The application during aseptic operations of strong antiseptics to bone in all probability causes necrosis of at any rate a thin, superficial stratum of bone tissue, but clinically nothing of this or of its results is ever seen unless infection has been added to the chemical trauma. Phosphorus necrosis of the jaw is another case in point; clinically the condition is not apparent until and unless infection becomes super-added.

In soft tissues bacterial action alone is not uncommonly the cause of an extensive gangrene—the result of the direct action on the tissues of a toxin whose virulence is out of all proportion to their normal powers of resistance. Yet even in such, experience has shown how often an abnormal lowering of local resistance by some severe vascular lesion

acts as an important accessory factor in the pathogeny of the gangrenous process. Necrosis of bone tissue in infective conditions would seem, however, to be much less often due solely to direct bacterial action than the result of a combination of this with a vascular disturbance—the extensive thrombosis and strangulation of vessels that so inevitably result from the occurrence of an acute inflammatory reaction within such rigid and unyielding walls. Hence the tissue reaction in acute osteomyelitis is not by any means a wholly beneficial process; it may be said to take place without regard to the structural peculiarity of the tissue which is its seat, and hence to over-reach itself and to result, not in defence, but in destruction. It is impossible, however, in any individual case to determine what part in the necrosis has been played by the direct toxic effect of the bacteria and what by the mechanical strangulation of vessels caused by the intensity of the defensive reaction.

Bacterial necrosis, then, occurs chiefly in acute infections in which the inflammatory action is severe, and much more readily in compact than in cancellous bone.

As we have seen, however, the superficial subperiosteal layer of bone receives its blood-supply largely if not solely from the vessels of the periosteum; this bone layer, then, will die on interruption of this supply through periosteal detachment. This is to be seen clinically in infective cases where periosteal stripping has resulted from the collection of pus in Macewen's "areolar tissue layer" and also after surgical or accidental detachment of the periosteum.

The Occurrence of Aseptic Necrosis.—Although almost the only form of necrosis that appears clinically is associated with, and in part at least due to, bacterial activity, yet it cannot be doubted that aseptic necrosis, exclusively the result of loss or impairment of blood-supply, does occur; a clear example is the death of bone in a limb that has become the seat of dry gangrene.

The point of greatest interest in connection with the occurrence of aseptic necrosis, however, is that of the viability of detached bone fragments in simple fractures and of the

bone-graft transplants of experimental and human surgery; whether these survive or undergo aseptic necrosis is not only of theoretical interest but of very great practical importance. The question of the fate of the former, the isolated bone fragments of simple fractures, is identical with that of the artificially detached transplants. Now it has been held that these transplants, whether experimental in animals or therapeutic in human surgery, not only do not take on active growth, but even that they do not survive; that they undergo necrosis, and as aseptic sequestra in part become absorbed, in part act as a mere passive scaffold for the building activity of invading osteoblasts. Thus Broca[1] writes: "The graft plays its part merely by exciting and directing an osteogenesis by substitution," *i.e.* it undergoes necrosis and subsequent absorption. If this be true, success in "taking" of the graft means merely that after necrosis substitution by new bone precedes the process of absorption of the aseptic sequestrum, failure on the other hand being due to this absorption taking place prematurely before the osteogenesis by substitution has had time to make good.

The view, however, that the occurrence of such necrosis is constant and hence inevitable is now pretty generally regarded as altogether too extreme. The observations and experiments of Macewen[2] furnish most convincing proof that bone tissue under favourable circumstances of transplantation is not only viable but capable of active growth. Bone bared of periosteum showed evidence of such growth as well as of absorption when transplanted into intermuscular planes and the peritoneal cavity; the growth of islands of bone cells from blood-borne osteoblasts implanted along the walls of an aneurysm; and finally, the evidence furnished by Dobrowolskaja[3] of the cultivation of bone tissue *in vitro* seem quite amply to justify Macewen's belief and expressed opinion that "under suitable surroundings the vegetative capacity of the

[1] Broca, *The After Effects of Wounds of the Bones and Joints*, 1918
[2] Macewen, *The Growth of Bone*, 1912.
[3] Dobrowolskaja, *Brit. Journal of Surgery*, vol. iv. No. 14.

osteoblast is at least as great as any of the tissues of the body and bone proliferation takes place from its whole surface circumferentially, while epithelium, for instance, proliferates only on the flat," and again " the vegetative capacity of the bone-cell is as great as that of the epithelial cell." Macewen himself owns, however, that in unfavourable circumstances this vegetative capacity fails and the bone then undergoes aseptic necrosis and absorption.

Albee [1] sums up the present position of knowledge in this respect well and fairly: " One should not be too dogmatic concerning the exact rôle that every graft must play. Individual conditions or individual environment determine the exact rôle of each particular graft, *i.e.* in favourable circumstances they wholly or in part survive and grow, in unfavourable die and fill merely a passive rôle." Such is probably true also of isolated fragments in simple fractures.

Caries is a destructive process occurring in all the varieties of osteomyelitis. Although in some acute cases rapid in its extension, it is essentially a gradual, progressive process, and, unlike necrosis, never one of sudden death *en bloc* of bone tissue. The phenomena of caries vary with the nature and the intensity of the irritant. Where this is an infective agent of a virulence sufficient to cause retrograde or degenerative changes in the bone-forming cells, caries results in destruction of all the elements of the bone; where, on the other hand, it is aseptic, or where infective of a subminimal level of intensity, the carious process consists of a mere rarefaction of bone, an absorption of the bone matrix only. Under such circumstances the irritant, of slight intensity, induces by its stimulant action proliferation of the cellular elements of the bone tissue.

Caries, an Acute Process resulting in Total Destruction of Bone.—Where caries is an acute infective process, the bacterial irritant is of a virulence out of all proportion to the resisting powers of the vital bone-forming cells, and its destructive effect is manifested by a localised necrosis of the

[1] Albee, *Bone-graft Surgery*, 1915.

bone and marrow cells plus a simultaneous progressive molecular disintegration of the matrix. The bone does not die, and becomes separated in macroscopic quantities, but in a succession of microscopic. The process is analogous to that of ulceration in soft parts; an analogy that holds good, however, only as regards the destruction of the organic elements of the bone; there is no phenomenon in ulceration comparable to the absorption of such inorganic substances as the bone-earths of the matrix. Whilst the direct toxic action of the bacteria is responsible for the death of the cells of the bone and marrow tissues, this absorption of the matrix is carried out by the osteoclastic cells which, prior to their death and destruction, assume an abnormally intensified activity. Possibly also a macerating and solvent action on the part of the inflammatory exudate assists this action of the osteoclasts.

Exactly what is the origin of these giant-cells is not very clear, but in appearance they are indistinguishable from the myeloplaxes of the marrow. Wherever inflammatory rarefaction or destruction of bone with absorption of its matrix is taking place the presence of osteoclasts is to be expected. Their activity in these circumstances is to be regarded as a renewal of a function that they are normally called on to perform during the growth and development of bone. As we saw in Chapter I, the development of osteoblastic activity with the laying down of bone in embryonic cartilage, the formation of medullary spaces, and in the long bones of the medullary canal are absolutely dependent on the absorptive action of these giant-cells (p. 6). In after-life, however, their function seems to remain latent, their powers being called out only in emergencies under the action of some abnormal irritant; this in acute destructive caries, while already of an intensity capable of destroying the essential formative bone-cells or osteoblasts, seems to act rather as a stimulant to osteoclastic proliferation and activity. Of course the mere fact of the occurrence of caries at all predicates the survival and activity of osteoclasts. Where these are destroyed prematurely before they

have completely fulfilled their function of matrix absorption necrosis, and not pure caries, takes place. Probably one reason why in compact bone necrosis and not caries is the usual result of an acute infective inflammation is that it contains too great a density and mass of bone-earths in proportion to the number of its osteoclasts to allow of the complete absorption of the former before the latter come under the paralysing and destructive action of the irritant. *Vice versa*, caries is *par excellence* the destructive inflammatory process of cancellous bone tissue partly at least because of the small relative amount of bone-earths it contains.

It is unknown how far these same cells are responsible for mere non-inflammatory rarefaction of bone, *e.g.* the rarefaction that occurs in disuse and in old age, in the causalgic form of nerve injury, and in osteomalacia.

The result, then, of caries in its acute infective form is the absolute loss or destruction of all the elements of bone tissue.

Caries, a Subacute Process resulting in Rarefaction of Bone Tissue.—Where, as in aseptic or subacute infective osteomyelitis, the irritant is of low intensity, stimulation and reactivation of osteoclasts with absorption and disintegration of the matrix is accompanied not by degenerative, but by proliferative changes in the bone cells or osteoblasts; indeed, as will be seen when we come to discuss the phenomena of bone repair, rarefaction of compact bone by osteoclastic activity is a constant accompaniment of all reparative proliferation of osteoblasts. Hence in these conditions caries results merely in a rarefaction of bone, a disappearance of the inert bone matrix, a process that is accompanied—although this is no part of the phenomenon of caries itself—by an increase in the osteoblastic cellular elements of the bone.

Of course, these two varieties of caries are in reality but two parts of one process, a focus of bone destruction from acute caries always being surrounded by a zone of bone rarefaction in which is simultaneously taking place a variable amount of osteoblastic proliferation.

Necrosis and caries are in several respects contrast processes, *e.g.*:

(1) Necrosis occurs most characteristically in acute inflammation of compact bone; caries in acute inflammation of cancellous bone.

(2) Where necrosis occurs in chronic disease it is rather in cancellous than in compact bone tissue; whilst destruction of bone by caries is a constant occurrence in the chronic process of separation of necrosed compact bone.

(3) Necrosis means the destruction only of the more vital parts of the bone tissue; caries in its acute form the absolute destruction and annihilation of all the elements of the bone; in its subacute form, absorption of the rigid matrix only.

Although so essentially different, the two processes are nevertheless closely connected; in the process of sequestration we shall see that the occurrence of necrosis is always followed by the action of caries. On the other hand, in tuberculous disease and also in some cases of septic osteomyelitis of cancellous bone necrosis may be the result of caries; a portion of the network of trabeculæ becomes absolutely isolated by the rapid extension around and beyond it of the carious process, and so cut off from its vascular supply and exposed to the direct action of the infection, undergoes death *en masse*.

The Occurrence of Caries.—Caries is most commonly and characteristically an infective process; less often does it result from the presence of an aseptic irritant. Examples of this, however, are to be seen in the rarefaction that occurs around metal plates and screws and other aseptic foreign bodies, and during the union of simple fractures and also in the rarefaction and destruction that result from the mechanical pressure of aneurysms and new growths. Associated with and due to the presence of an infective irritant, caries is to be seen most typically—

(1) In acute infective inflammation of cancellous bone.

(2) In chronic infective inflammation of cancellous bone. A typical example is the gradual excavation of an epiphysis or short bone like the os calcis, in which a septic foreign body has been allowed to remain embedded. *Vide* Plates XVII. and XVIII., facing p. 128. The formation of an abscess cavity in "Brodie's cold abscess of the tibia" and in post-typhoidal osteitis are further examples.

(3) In the chronic granulomata occurring in cancellous bone, *e.g.* in tuberculosis, syphilis, and actinomycosis.

(4) In all conditions where necrosis has taken place and the dead bone retains continuity with living bone tissue. Thus caries is universally present and the responsible agent in the process of sequestration.

The Results of Caries.—Caries, though a destructive process, is no more an unmitigated evil than ulceration; under strictly analogous conditions it has corresponding advantageous results.

The Separation of Necrosed Bone.—In like manner as the process of ulceration achieves the liberation of sloughs without waiting for their gradual disintegration, so the process of caries effects the separation of masses of dead bone, whose slow absorption or disintegration by the tissues is impossible.

The Phenomena of Sequestration.—This process of sequestration is the most beneficial of all the results of caries; it constitutes the first and the most definitely purposive of the steps in the elimination of dead bone; although the time required for its completion necessarily varies greatly according to the different circumstances of individual cases, the liberation always eventually becomes complete; on the other hand, it is quite exceptional for further stages in the spontaneous elimination of sequestra
h completion.
radual solution of continuity between the living a

the dead, which is the whole process of sequestration, takes place by disintegration of the living bone tissue; the liberation of dead bone is always performed at the expense of the living. This, of course, corresponds exactly with what occurs in ulceration.

A certain area and depth of bone has undergone necrosis. This bone, dead and moreover a focus of chronic but ever-present infection, acts as a continuous irritant to the living bone tissue in its immediate neighbourhood which consequently to a variable depth and over a variable width becomes the seat of a gradual destructive process; where adequate drainage has been provided and maintained the inflammatory reaction induced in this living bone is not usually sufficiently severe nor the action of the bacterial toxins virulent enough to cause actual necrosis in this zone of bone destruction. The process of caries alone is responsible for the erosion and annihilation of the living tissue over a variable width and to the full depth of the original necrosis. But liberation of the dead mass by caries is not of course the only process taking place during this period. The same irritant responsible for the destructive phenomenon has been calling into existence defensive and reparative changes by its stimulating action on the bone-cells of the tissue that lies beyond the range of its destructive influence. Such are manifested in the first place by the appearance on the edge of the carious process of a granulation tissue that in many cases, with the prolongation of the period of sequestration, becomes of so exuberant a growth as to overlap and partly to conceal the as yet incompletely separated dead bone. The simultaneous occurrence of a second reparative phenomenon, that of sub-periosteal osteogenesis, too often proves an effective barrier, not indeed to the liberation of the dead bone, but to its further elimination as a sequestrum from the body.

The duration of the process of sequestration, the time that elapses before the carious process has destroyed all bony continuity between the living and the dead, varies with the intensity of the inflammation, with the density of structure,

and the thickness of the cross-section of the bone at the line of separation. Hence on the average it is shorter in cases of the separation of superficial lamellæ, and of dead masses of cancellous tissue; longer where the whole thickness of a shaft of a long bone has to be eaten through, longest of all when such a bone is the femur. The actual period for superficial lamellæ is usually from fourteen to twenty-one days; for the complete cross-section of the shafts of such bones as the fibula, the radius, and the ulna from three to four weeks; in the case of the tibia about three months; whilst in the case of the femoral shaft from three to four or even six months. Cases are met with in which the process takes even longer periods for its completion; but these are exceptional.

The Varieties of Sequestra in Traumatic Osteomyelitis and their distinguishing Characteristics.—Apart altogether from the division of sequestra into aseptic and infected, there exist definite varieties of the latter that are distinct in their pathogeny and their physical characteristics. Necrosed bone becomes a sequestrum whenever or as soon as it is loose, and completely detached on all sides from neighbouring live tissue. In compound fractures, however, many splinters of bone become so detached and isolated by the original trauma. In such their necrosis follows their isolation; no process of sequestration is necessary or takes place. These retain their original shape, and are called early primary sequestra. Later primary sequestra are those formed by fragments of bone that, detached by the violence of the original injury from all but a limited connection through the periosteum with soft parts, and so undergoing early necrosis, rapidly become wholly detached and completely isolated by ulceration of their limited band of periosteal attachment. Of course every such piece of partially detached bone does not undergo total necrosis; some retain sufficient blood-supply to resist the infection; others enough to result in survival of a part of their mass. Both varieties of primary sequestra are readily distinguishable by the characters of their borders, all of which, repre-

senting the original lines of traumatic cleavage, although often sharp, remain relatively smooth and plane.

Secondary sequestra represent bone that, unaffected as regards its blood-supply by the original injury, but dying during the subsequent stage of acute inflammation, still when dead has retained physical continuity with the living bone until set free along the line of this previous continuity by the process of caries. In traumatic osteomyelitis such sequestra may be said to form the second crop; they are easily distinguished by the fact that whilst certain of their borders represent the original lines of splintering of the bone, they always present at least one border that, distinctly jagged and serrated, betrays the fact of its gradual formation and separation. Such serration, the result in the first place of irregularity of depth and extent of involvement of the bone in the process of necrosis, is absolutely characteristic of any bone surface or edge that has been gradually defined by caries during the process of sequestration.

A third type of sequestrum exists, much commoner in the spontaneous osteomyelitis of childhood than in the traumatic form, and yet not infrequent in cases of the latter disease in which, with late exacerbations of the infection and inflammation, there has taken place necrosis of bone, other than that originally involved in the trauma. When such bone comes to form a sequestrum all its borders and even all its surfaces will show the serrated appearance we have just described, none of them will have the clean-cut sharpness of definition that results from participation in the original splintering and comminution. In traumatic infective osteomyelitis such sequestra may be termed tertiary.

Changes that take place in Necrosed Bone and Sequestra.—At any rate in the presence of infection necrosed bone and sequestra undergo a definite change in composition due to the rapid disappearance of the cellular elements of the bone tissue proper, and of the marrow as well as of the blood-vessels, nerves, and fatty and other connective tissues they contain. The disintegration and disappearance of these is the result partly of phagocytosis, partly of bacterial action,

and partly no doubt of the activity of autolytic ferments. The empty spaces thus created become filled with air and pus. Does the other element of the bone—the matrix—undergo any change? From a comparison of the results of analysis by decalcification of normal bone with those of a series of sequestra of compact bone, J. G. Greenfield and the writer have been able to prove that no disappearance by absorption or disintegration of these takes place no matter how long the dead bone lies bathed in pus and the body fluids. In these investigations the percentage by weight of bone earths in compact bone, as distinct from the total organic material, did not exceed 68·9%, this figure representing the inorganic content of the dense bone of the tibial crest. Compare with this figure the following:

(1) The bone-earth content of a sequestrum of the shaft of the humerus removed five months after the occurrence of necrosis—70%.
(2) The bone-earth content of a great number of large sequestra of the shaft of the femur removed twelve months after their necrosis—70·45%.
(3) The bone-earth content of sequestra from the shaft of the femur removed five months after necrosis—71·2%.
(4) The bone-earth content of sequestra removed from the shaft of the femur six months after their necrosis—67·45%.

The general average of the bone-earth contents of these sequestra of compact bone was thus found to be 70%. This increase in percentage over the normal almost certainly represents the loss of the cellular elements that we have described, and proves that there has been no loss of the inorganic elements of the matrix; whilst the slightness of the increase at least suggests that there can have taken place little or no disappearance of the organic matter of the matrix.

Sequestra, then, consist merely of the matrix of bone tissue, ossein plus the bone-earths. The disappearance

from necrosed bone of the other elements and their replacement by air result in two absolutely characteristic clinical features: the former gives rise to the grating sensation imparted to the probe, the latter to the resonant note elicited on percussion with a probe.

The Ultimate Fate of Sequestra.—As we have seen, aseptic sequestra seem in part to undergo absorption, in part to act as a passive basis for further osteogenesis by invading cells.

Septic sequestra, on the other hand, as the figures just quoted prove, are not absorbed. They have, however, a natural tendency to elimination by spontaneous emergence and discharge through the soft parts surrounding them, a tendency that is, however, in the majority of cases effectively thwarted by the reparative osteogenesis that has taken place around and so as to enclose them. Where, however, a sequestrum has not been thus encased in a sequestral cavity, or where, although encased, it is of such a shape and size as to permit of its ready escape through the natural openings of such a cavity, its spontaneous emergence through the soft tissues either by passing along an existing sinus or by ulcerating a new path for itself, will probably sooner or later take place. In other rarer cases the spontaneous emergence of encased sequestra takes place by an extension of the carious process around them, which comes to involve a free surface of the bone; the sequestra in such cases may be said to eat their way out through the bone, and then, by a further and analogous process of ulceration, make their way through the soft tissues and so eventually are discharged.

This may be regarded as the second of the beneficial effects of caries—its occasionally resulting in the spontaneous emergence of encased sequestra. Such an occurrence is, however, comparatively rare; in the great majority of cases the conditions necessary for its accomplishment do not obtain, whilst in the few cases in which they do obtain it is always a long-delayed, uncertain, and more or less accidental and hap-hazard occurrence, and one that necessarily

implies a further destruction of bone tissue with a corresponding increase in the size of the already existing bone cavity. Hence it is by no means wholly a beneficial process.

Failing their surgical removal, then, the great majority of sequestra in chronic traumatic osteomyelitis will remain enclosed within their sequestral cavities for absolutely indefinite periods.

The Evil Results of Caries.—So far we have been dealing with the results of caries that, as regards the final healing and cure of the diseased process, are in some respects at least beneficial; but of course caries, like ulceration, is an essentially destructive process. Its primary and most characteristic effect is that of loss or destruction of bone tissue—an effect that in many cases renders healing difficult and even impossible.

Its extension with involvement of a free non-articular surface of the bone at one or more points ends in the appearance of multiple and successive abscesses with consequent formation of chronic sinuses that inflict permanent damage on skin and muscle; whilst its extension with involvement of articular surfaces has more serious results—the occurrence of one or other variety of septic arthritis, an event that is most likely to occur early in the disease during the first acute stage of the infection, but which may occur at any later period on the occasion of an acute exacerbation of the infection or of a more gradual and less noticeable local extension of the carious process.

The evil result of caries, however, that in cases of traumatic infection of masses of cancellous bone constitutes the greatest obstacle to spontaneous healing is **cavity-formation.**

The Formation of Cavities in Bone

As regards cavities in bone, caries is not by any means the only factor or pathological process concerned in their production. Moreover, the etiological factors of their initiation, the pathological processes that result in their development and extension, and the presence or absence of

infection, are all considerations of the first theoretical and practical importance. Hence such cavities may be classified from each of these three points of view: the etiological, the pathological, and the bacteriological.

Classification of Bone Cavities on an Etiological and Bacteriological Basis.

(1) Aseptic cavities of traumatic origin.
  (*a*) The aseptic cavities of simple fractures.
  (*b*) The cavities left after the removal of an aseptic foreign body.
  (*c*) The cavities left after the removal of new growths of bone.
(2) Aseptic cavities of spontaneous origin.
  (*a*) The cavities due to new growths.
  (*b*) The cavities of fibro-cystic disease of bone.
(3) Infected cavities of traumatic origin.
  (*a*) Cavities the result of the presence of an infected foreign body in bone.
  (*b*) The cavities of compound fractures proper, either before or after the performance of simple sequestrectomy.
(4) Infected cavities of spontaneous origin.
  (*a*) Cavities the result of chronic spontaneous (hæmatogenous) osteomyelitis.
  (*b*) Cavities the result of acute spontaneous (hæmatogenous) osteomyelitis.

Of course, any aseptic cavity may become, either after surgical interference or spontaneously by hæmatogenous infection, an infected cavity.

As regards the pathological processes that take part in the formation of each of these varieties of cavity, these will be dealt with in the next chapter. It will be sufficient here to state that direct trauma, caries, necrosis, osteosclerosis, and osteogenesis, singly or in combination, are concerned in their production.

The Reparative Phenomena of Osteomyelitis

General Consideration of the Phenomena of Tissue Repair.—Repair is to be considered as part of the tissue reaction that constitutes inflammation. It is the result of the sum total of the reparative or regenerative elements and phenomena of inflammation. Even aseptic repair must be regarded as an integral part of the reaction of tissue in the presence of an aseptic irritant; the aseptic healing of a clean-cut wound, *e.g.* of a "clean" surgical operation wound, takes place solely by inflammatory reaction on the part of the damaged tissues, no matter how apparently slight the damage they have been subjected to or the irritation thereby induced.

In the higher animals, repair is to a very large extent effected by the proliferative activity of connective tissue cells; in the presence of injury or destruction most of the other more highly differentiated tissues are in this matter of repair subserved by the ever-ready productivity of the connective tissues. Of these more specialised tissues, the epithelial cells of certain glands and lining membranes retain to the greatest extent the power of replacement of loss by their own proliferation, and are thus able to play a supplementary part in the repair of the destructive effects of injury or infection.

Hence the presence in the tissues of an irritant is one essential condition of the occurrence of repair. Such irritant will, however, be as diverse in its nature and mode of action as those responsible for the destructive phenomena in an area of inflammation. There is, however, a definite relation between the intensity of the irritant and its action in tissue repair. Where this intensity becomes increased beyond a certain point a zone of negative chemiotaxis is induced in the tissues immediately surrounding it—a zone in which destructive change soon becomes apparent. Outside this there almost always exists a further zone of induced positive chemiotaxis, the result of a diminution in the intensity of the irritant proportional to the distance through

which it is acting. Where, on the other hand, its intensity falls below the minimal level necessary for the induction of negative chemiotaxis, it acts on the tissues in its immediate neighbourhood as a stimulant, a producer of regenerative or defensive changes. In either circumstance, then, whilst acting on tissue either contiguous or at a distance, it is by a positively chemiotactic influence, as a stimulant of cellular activity, that the irritant initiates the process of repair and effects the production of new tissue.

The appearance in intimate relation to the dilated capillaries of numerous new spindle-shaped cells of fibroblasts is the earliest sign of this tissue stimulation, and marks the first phase in the process of repair. The question of the origin of these fibroblasts has not as yet been finally settled; most, if not all, arise from the pre-existing fixed cells of the connective tissue and from certain endothelial cells of local origin, possibly some from certain large mononuclear cells of doubtful life-history. Besides fibroblasts and these their known or possible parent cells, polymorphonuclear and eosinophil leucocytes are to be found in this new granulation tissue. In acute inflammation, granulation tissue makes its appearance, then peripherally and at a distance from the focus and centre of irritation, and represents there the stimulant action of an irritant weakened by distance. With subsequent diminution of intensity of this irritant further growth of granulations takes place towards it; the progress of repair being thus centripetal, towards the centre of inflammation.

Simultaneously with this progressive growth of granulation tissue, there takes place in the earlier formation—whether by active secretion or by passive conversion of the fibroblasts is still disputed—a development to scar tissue, the richly cellular tissue changing into a dense matrix of white connective tissue fibres, poor in cellular elements.

Just as the initiation of the process of repair takes place in a vascular area, so the continuation of cell proliferation with consequent formation of granulation tissue demands an adequate supply of nutrition. Such growth, then, is

always accompanied, in part preceded by, the formation of loops of new capillary vessels, the latest formed layer of granulation being thus always exceedingly vascular.

The progressive centripetal conversion of granulation tissue into scar tissue, with the subsequent gradual contraction of the latter, entails certain vascular results of far-reaching practical importance. At first scar tissue remains highly vascular; but in time its contraction reduces both the number and the size of the vessels passing through it, so that while young scar tissue is always vascular, old scar tissue becomes less and less so, in the end almost completely avascular. The practical significance and importance of this is twofold. In the first place, old scar tissue, being hard and dense and so ill supplied with vessels, is in a position to resist but poorly any sudden exacerbation of the irritation that has produced it; it is incapable of becoming the seat of an acute inflammatory reaction; owing to its density and consistence, such swelling and exudation as occur but further interfere with what circulation there is; hence it falls an easy prey to the destructive action of an infective irritant and is only too prone to undergo massive necrosis, or sloughing. On the other hand, the progressive contraction of earlier formed scar tissue at the periphery of a damaged area, resulting as it does in an increasing paucity of blood-supply through it, is at the present time an only too common cause of failure of completion of the natural spontaneous repair in large wounds.

The Various Modes of Repair in Soft Parts

(1) **Repair by First Intention.**—In a clean-cut wound the process of repair, though not necessarily sterile, is yet aseptic; any germs that have been carried into the tissues fail to develop, being destroyed by phagocytosis and bacteriolysis. Tissue reaction to the mechanical irritant of the injury gives rise to a thin layer of granulation tissue that replaces a corresponding layer of blood-clot, itself in turn becoming converted into red vascular and later into

white avascular scar tissue. At the same time replacement of destroyed and damaged cells of the epithelial layer of skin or mucous membrane is effected by proliferation of its neighbouring sound cells.

(2) **Repair by Granulation.**—Although in some cases this may commence as an aseptic process, the occurrence of infection early or late is almost inevitable, whilst in most cases micro-organisms have been present from the beginning. Hence this is, *par excellence*, the method of repair in all suppurating wounds, in all cases where tissue loss has been caused or complicated by the presence of infection. The micro-organisms present, in whatever numbers and in whatever variety, constitute the irritant; simultaneously with regeneration and ingrowth of epithelial cells, granulation tissue continues to form and develop and replace the tissue loss; a large hard vascular scar results, giving way in time to one white, soft, of less prominence and greater suppleness.

(3) **Healing by Secondary Adhesion.**—This is really a variety of repair by granulation. When two granulating surfaces meet, whether spontaneously by mutual collapse or gradual growth of granulations, or brought and kept in contact by surgical means, healing takes place by secondary adhesion of the two. This is the mode of healing that obtains in successful cases of secondary suture of infected wounds. The surfaces, however, are neither strictly aseptic nor perfectly regular. Hence irregular small pockets and recesses containing germs often remain for a variable time sealed off by the union of granulations around them. In most cases such germs soon fall victims to the combined forces of phagocytosis and bacteriolysis; but in war wounds they often survive in such recesses for indefinite periods, and with most evil potentialities for spontaneous or post-operative recrudescence of their activity.

(4) **Replacement Fibrosis.**—This is the name given by Adami to the process of gradual replacement by granulation and scar tissue of abnormal masses of dead or inert absorbable material. The commonest example of this is the

gradual conversion of a hæmatoma from without inwards into scar tissue.

Similar and corresponding processes occurring in the repair of bone tissue bear a strict analogy to each of these four methods of repair in soft parts.

Incompleteness of Natural Repair in Soft Parts

Failure of completeness in natural spontaneous repair of tissue loss may be due to one or any combination of the following causes:

(1) **The persistence in the tissues of an irritant which they are incapable of removing,** *e.g.* a splinter of dead bone or a septic foreign body, or of destroying, as in the chronic tubercular or granulomatous conditions, in which a balance is struck between the resisting power of the tissues and the aggressive destructive power of the micro-organisms, a balance that may remain stationary over long periods.

(2) **Failure of Healing by Granulation.**—This is a very common occurrence in the extensive wounds of the present time, and is due to contraction of the earliest formed peripheral layers of scar tissue taking place before healing is complete in the centre of the wound, a process that leads by premature cutting off of adequate blood-supply to the innermost layer of granulation tissue, to cessation of healing by granulation, and, since epithelial regeneration is also dependent on the blood-supply to the granulation tissue, to a similar cessation of epithelial ingrowth. In this manner arises the very common chronic ulcer or indolent wound, with its central bluish-red area of granulation, its hard peripheral ring, and fixed rigid base of dense scar tissue.

(3) **Failure of Healing by Secondary Adhesion.**—Inability on the part of the walls of a cavity to collapse because of local mechanical or anatomical conditions renders healing of the cavity by this method impossible, unless the growth of granulations on one or both walls prove so rapid and exuberant as to enable them to meet and adhere before their blood-supply is interfered with by contraction of the peri-

pheral scar-tissue. Such spontaneous obliteration is possible only in small rigid cavities where the distance to be bridged by granulation is not great. The classical example of the hopelessness of Nature's efforts at the healing and obliteration of a large cavity with rigid walls is seen in chronic empyema; one wall, the parietal, is held rigid by the bony framework of the thoracic cage, the other, the collapsed lung with its visceral covering of pleura, has long since in the early days of the disease become firmly fixed and inexpansible by adhesions and fibrosis. Cure can be brought about only by such removal of the rigid bone elements from the chest wall as will allow the collapse of this wall into the cavity, into contact with the inner. Such is the aim of the operations of Estlander and Scheede. Examples of a similar condition are to be found in abscess cavities that lie between two bones or between a taut tendon and a bone.

(4) **Failure of Completeness of Replacement Fibrosis.**—On cutting through the dense scar tissue that represents the organisation of a hæmatoma, it is not uncommon for the surgeon to come across pockets of clot still unabsorbed or replaced. The cause for such failure of completeness in replacement fibrosis is again to be referred to the cutting off of blood-supply to the central layer of granulation before the process of organisation of the whole clot is complete. Although some of the irritant still remains, further attempt at its complete suppression has been effectually stopped by inadequacy of nutrition.

All these conditions of incomplete healing in soft parts have also their exact counterparts in conditions that arise in the repair of lesions of bone tissue.

Repair in Bone Tissue

Bone is a connective tissue, and the general principles of the process of repair in bone are identical with those we have just outlined as obtaining in soft parts, proliferation of connective-tissue cells occurring in response to the stimulation of an irritant in the presence of an adequate blood-supply.

The bone cell, however, is a specially differentiated connective-tissue cell; when, therefore, as an osteoblast it reverts to a condition of embryonic activity it comes to repossess a more specialised function than does the fibroblast. Activity of this latter cell converts granulation tissue into ordinary scar tissue; whereas the activity of the osteoblast—the unit of granulation tissue of bone—results in the appearance of callus, which is nothing more than young vascular scar tissue with osteoblasts in the place of fibroblasts and a calcified matrix instead of ordinary connective tissue fibres. Just as in scar tissue there is a progressive increase in density with a corresponding decrease in vascularity, so this vascular callus becomes gradually converted into bone scar or osteosclerotic tissue, with few vessels but with a dense homogeneous bone matrix. The process of formation of such bone-scar is known as osteosclerosis; as, however, in many cases its production is greatly in excess of the amount of bone tissue lost, it is often referred to as an osteogenesis.

The rôle played by cartilage cells in the repair of bone tissue is not well understood. As we have seen, Macewen regards the osteoblast as primarily a derivative of the chondroblast, or embryonic cartilage cell; several writers claim that the formation of cartilage is a constant stage in the repair of simple fractures of human bones. Certainly this is so in the bones of some animals; certainly it occurs too in some cases of ununited fractures of human bones.

As regards their ultimate fate, ordinary scar tissue and sclerotic bone present an important point of contrast; the former shrinks and contracts and becomes progressively poorer in blood-vessels, whilst bone scar, especially where formed under aseptic conditions, often undergoes a late remodelling of structure, remaining dense in accordance with and along the lines of crystallisation of the forces transmitted through it, elsewhere becoming rarefied or entirely absorbed by osteoclastic activity. In time, moreover, it develops the structure of normal bone with Haversian systems.

There is, moreover, a further point of difference between the two reparative processes; the rigid structure of compact bone has no analogy in soft tissues; it affords but little space or room for increase of vascularity, practically none at all for cell proliferation. This results in the early accumulation of osteoblasts beneath the periosteum, the loose areolar layer of which easily accommodates them. It also explains the necessity for the constant association of rarefaction with reparative effort in compact bone tissue. Stimulation with proliferation of osteoblasts can hardly take place unless simultaneous compact stimulation of osteoclastic activity results in rarefaction of the compact bone.

Yet, on the whole, the general principles of the process of bone repair are so closely akin to those of repair in soft tissues that one might on *a priori* grounds be justified in expecting as frequent a complete and adequate repair of loss or destruction of bone as takes place in other connective tissues. In repair of bone it becomes necessary to draw a distinction that in the case of the soft parts does not obtain to anything like the same extent. The pathological process of regeneration of bone tissue varies greatly as it is septic or aseptic, according as the irritant responsible for it is an infective or a non-infective agent. Not only from the theoretical but from the practical standpoint the question of the presence or absence of infection dominates all others; on it depend not only differences in pathology, but necessarily and on that account differences in clinical history, prognosis, and treatment. Aseptic repair of bone may be regarded as the normal process of true bone regeneration, bone tissue being replaced by bone tissue; repair in the presence of infection, on the contrary, not only is very often atypical, bone tissue being replaced in part at least by ordinary undifferentiated scar tissue, but in very many cases remains for various reasons incapable of spontaneous completion.

Modes of Repair in Bone Tissue

The Aseptic Repair of Bone.—Like the similar process in soft parts, aseptic repair of bone is to be considered as essentially inflammatory in nature, the result of a reaction of tissue in the presence of an aseptic irritant. Hence we must regard and accept cases of simple fractures in the process of uniting as cases of aseptic osteomyelitis.

There are two modes of aseptic repair in bone; these correspond respectively to the healing by first intention of a clean-cut wound of soft parts, and to the process of replacement fibrosis.

The former, which is really repair by first intention taking place in a specialised connective tissue, may be best studied, as Ollier studied it, in the union of a clean saw-cut through the shaft of a bone as far as the medullary canal. A thin line of bone tissue is in this way destroyed; the linear dead space thus formed fills with blood-clot, which together with the original trauma acts as an irritant and induces in the bone tissue of its immediate neighbourhood an increase in vascularity with a revival of embryonic osteoblastic and osteoclastic activity. Proliferation and outpouring of bone cells taking place concurrently with absorption of bone matrix results not only in the conversion into granulation tissues of a thin layer of the bone on either side of the saw-cut, but in replacement by granulation tissue of the blood-clot itself. The osteoblastic cells of this granulation tissue secrete a calcified matrix—hence the formation of callus or young vascular bone-scar tissue. With progression of the osteosclerotic process this in time becomes decreasingly vascular and increasingly dense. Such osteoblasts as remain ultimately resume adult characteristics and become ordinary bone cells again. Later comes the reconstruction process, already described, with the exception of which all the stages in this mode of union or repair correspond exactly with those in the repair of a clean-cut operation wound. Union by such a method obtains also in certain simple subperiosteal fractures—mere cracks and fissures of bone—without dis-

placement of fragments or detachment of periosteum, in which the bone loss consists of no more than a mere line. It is moreover by a process, identical or at any rate essentially similar, that union takes place between a well-fitting bone transplant and its bone host.

In the majority of simple fractures, however, even in those without displacement, a varying amount of periosteal detachment with subperiosteal and intramedullary hæmorrhage takes place ; repair and union are brought about in these circumstances by a process that is analogous to the replacement fibrosis of Adami. This same mode of repair obtains in all aseptic bone cavities, *e.g.* in the cavities that remain after the removal of an aseptic foreign body or new growth. Indeed from the point of view of tissue repair, all such simple fractures as we are here dealing with, all those with displacement or periosteal detachment, are to be regarded as cases of more or less complicated aseptic traumatic bone cavities : in all there exists a dead space variable in size and shape that becomes filled with blood-clot. The lining wall of such aseptic bone cavities of fractures consists peripherally of the sheath of detached periosteum, centrally of bone surfaces, *viz.* the external from which the periosteum has been stripped, the intermediate fractured surfaces and the inner walls of as much of the medullary canal as has been opened up. *Vide* Diagram 1.

In a typical case of diaphyseal fracture without displacement, comminution, or actual periosteal laceration, such a cavity remains closed, and may be regarded as consisting of three concentric zones—an outer zone, lying between the detached periosteum and the bone surface from which this has been torn, an intermediate or middle zone corresponding to the narrow space that exists between the actual fractured surfaces of the compact bone, and an innermost central area made up of a small portion of the medullary canal of each fragment and of the space connecting these. *Vide* Diagram 1 (p. 36).

By the usual process of aseptic repair in bone that has been already described each of these three concentric zones

of blood-clot becomes in turn a zone of "bone" granulation tissue, formed of osteoblasts, a mass of callus or young vascular "bone" scar, and finally dense osteosclerotic tissue or old "bone" scar: hence arise the external, the intermediate, and the internal callus, and external, the intermediate, and the internal masses of "bone" scar, the whole the result of the proliferative and secretory activity of osteoblasts.

Concurrently with the proliferation of osteoblasts in the

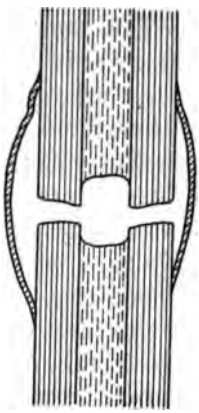


DIAGRAM 1.—The three zones of the fracture cavity in a simple fracture with neither displacement nor comminution.

original bone tissue there takes place, by a process of subacute aseptic caries, that rarefaction and decrease in the relative amount of rigid matrix that we have already noted.

The origin of the osteoblasts in each of the three different zones, but particularly in the outer or subperiosteal zone, has been the subject of investigation and of a controversy that has centred round the question of the periosteal origin of the external callus—the provisional callus of Dupuytren—which, as we shall see, is by far the most important factor in the union in all fractures with displacement. Until lately the classical experiments and the authoritative opinion of Ollier had resulted in a general belief in the inherent osteo-

genetic powers of the periosteum as displayed in the repair of simple fractures and of septic osteomyelitis alike. That the periosteum does possess powers of bone formation remains true, but the experimental work of Macewen and others showing the result of careful aseptic subperiosteal resection of bone, of transplantation of periosteum, and also the effect on bone growth of complete removal of periosteum has proved conclusively that the osteogenetic properties that it seems to possess are in fact not its own, but due to a resumption of osteoblastic activity of the cells of minute portions of bone that have adhered to it, having been detached with it. How far in simple fractures this external callus results from activity of the osteoblasts of the bone itself and how far from the activity of those detached with the periosteum and subsequently nourished by it still remains a controversial point. Macewen considers that in the great majority of cases the formation of external callus is almost wholly independent of the periosteum. In simple fractures certainly from a practical point of view it would not seem to matter greatly in what proportion of cases the general trend of growth and development of external callus and bone scar is from within outwards and in what proportion in the opposite direction from periosteum to bone. In compound fractures, however, and especially in infected gunshot fractures, it becomes a point of great practical importance. It is a question that we will discuss more fully in considering the repair of infected fractures.

With the occurrence in different cases of displacement and multiplicity of fragments and of more or less extensive periosteal laceration not only does the whole cavity undergo innumerable changes of shape and size, but the relations of its three zones becomes altered. Reference to Diagrams 2, 3, 4, and 5 will make this clear at a glance ; thus in Diagrams 2, 3, and 5 the intramedullary canal spaces communicate directly with and really form part of the outer or subperiosteal zone, whilst in Diagrams 3 and 5, where complete overlapping of the fragments has occurred, the second intermediate zone has been eliminated, and the subperiosteal outer zone has

now come to include all three; in Diagram 4, where comminution but little displacement has taken place, the intermediate or middle zone has become exceedingly complicated. In Diagram 5 periosteal laceration complicates the picture,

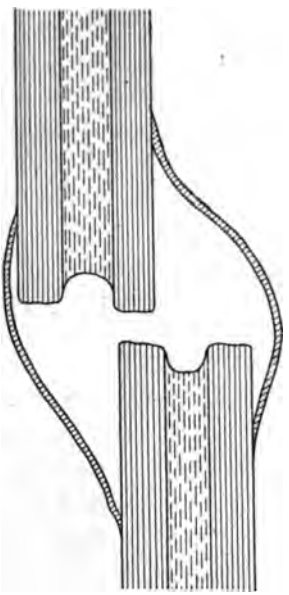


DIAGRAM 2.—The fracture cavity of a simple fracture without comminution but with considerable lateral displacement. The internal zone now opens into the subperiosteal, the intermediate zone exists at one point only.

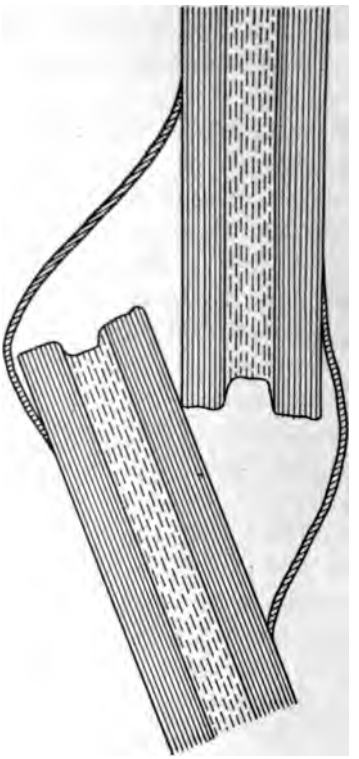


DIAGRAM 3.—The fracture cavity of a simple fracture without comminution but with overlapping of the fragments. All three zones are now merged into one, the external or subperiosteal.

and the hitherto closed cavity now communicates and becomes one with those of intermuscular and subcutaneous hæmatomata.

The greater the amount of the displacement, then, the more important factor in the union does the subperiosteal callus

become; but as long as the periosteal sheath is intact it limits on the outer side the growth of the osteoblasts. Where, however, this is torn—and frequently in such cases an end of one or more of the fragments projects out through the tear—osteoblastic outgrowth is no longer so limited, but,

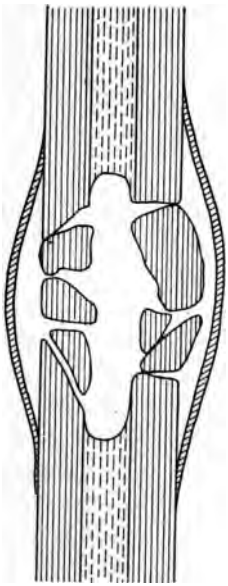


DIAGRAM 4.—The three zones of the fracture cavity of a simple fracture with comminution but with little or no displacement of fragments; the internal zone is of considerable extent, the intermediate has become very complicated.

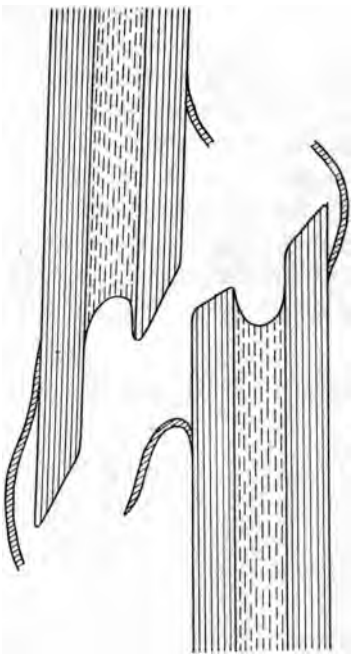


DIAGRAM 5.—The fracture cavity of a simple fracture without comminution but with marked displacement and associated with periosteal laceration; all three zones are merged into one, and this communicates with the tissues that are extraperiosteal.

replacing blood-clot, it invades extra-periosteal tissues, chiefly muscles. In all fractures, but especially in such cases of extra-periosteal callus formation, the amount of external callus is limited and its shape moulded by external pressure—in itself constituting, as Macewen has pointed out,

a very strong reason why fractures of the lower end of the humerus should be treated in extreme flexion.

In all complete diaphyseal fractures the reconstruction and remodelling of bone scar is of the greatest importance. Where no displacement has occurred the internal and external masses of scar become largely absorbed with re-establishment of the original conformation of the bone, and of the continuity of the medullary canal, whilst the intermediate mass also in time develops an Haversian system. Where permanent displacement of fragments has taken place, not only the bone scar, but also the original bone tissue undergoes correspondingly extensive changes in shape and structure, adapting itself, here by absorption or decrease in density, there by thickening and increased density, to the changes in direction of the lines of force it has to transmit consequent on the alteration in the axes of the bone.

Repair of Bone Tissue in the Presence of Infection, in Infective Osteomyelitis

The fundamental phenomena of bone repair in the presence of sepsis bear much the same relation to those of aseptic bone repair as the healing by granulation of an infected hæmatoma does to the replacement fibrosis of an aseptic one. The blood-clot effused into the seat or cavity of the fracture is not gradually replaced by osteoblastic granulations, but becomes rapidly disintegrated by the action of the bacteria; granulation tissue is slow in making its appearance. From this point onwards, however, the phenomena are the same as in aseptic repair, except that there is a very great tendency to failure in completeness of the process as regards both the occurrence of final healing and the degree to which bone tissue is replaced by bone-scar tissue.

Just as there are two modes of repair of infected wounds of soft parts, so are there two corresponding methods of repair in infective osteomyelitis.

The former of these is analogous to the healing by granulation of soft parts. Such is the method that obtains on

bone surfaces and in shallow superficial infected cavities—"open" cavities that possess a slightly concave floor but no definite walls and no roof. A typical example of this mode of healing by granulation is to be seen in the very common shallow cavity of the subcutaneous surface of the tibia such as results from an incomplete fracture, the result of a kick from a horse or a "gutter" shell wound.

The skin and soft parts, with a variable amount of bone and periosteum, have disappeared, either carried away by the original violence or destroyed by the subsequent infection; necrosed bone has separated and the sequestrum been removed; a shallow cavity remains. During the process of sequestration, however, the first stages of repair have been taking place. The bone forming the floor of the cavity, vascularised, rarefied, in part destroyed by the irritant action of the infected sequestrum, has at the same time been stimulated to proliferation and outpouring of osteoblasts, and by the time the separation of the dead bone is complete it is already carpeted by granulations. Growth of these goes on until the cavity is more or less completely filled, when healing takes place by epithelial ingrowth. The granulation tissue subsequently passes through the ordinary stages of development into bone—a bone scar results covered by a mere thin epithelial layer. A similar series of changes may be watched by the naked eye in the infected bone end of an amputation stump. Simultaneously with the destructive changes of sequestration there take place the proliferation and outpouring of osteblasts; growth and development of the resulting granulation tissue coincide with proliferation and ingrowth of epithelial cells; a bone scar covered by the common circular adherent cutaneous scar is the result.

The second of the modes of repair in infective osteomyelitis is really identical with the process of healing by secondary adhesion of granulating walls that has been described as taking place in abscess cavities with collapsible walls. This is the mode of repair in all septic bone cavities—secondary adhesion of one granulating wall with another

as of the bone ends of a diaphyseal fracture with but little displacement or bone loss, or by secondary adhesion, spontaneous or post-operative, of one granulating bone wall with a granulating surface of soft tissues. In all such closed infected cavities as are not quite small, the occurrence of complete spontaneous healing is, however, very exceptional. It is only where a narrow space has to be bridged over by outgrowths of granulations from the walls of the cavity that spontaneous healing is likely to occur.

Incompleteness of Natural Repair in Osteomyelitis

Complete repair in osteomyelitis implies the fulfilment of two conditions:

(1) The restoration of bone tissue that has been lost; this includes in diaphyseal fractures the most important desideratum of sound bony union.
(2) Where infection is present, the complete and final healing of the septic focus.

Failure in Aseptic Repair.—This means, then, failure of replacement of bone loss. The most important example of this is non-union of a simple fracture. The causes of this are numerous, but fall naturally into three groups:

(1) Conditions interfering with the nutrition of the osteoblast.
(2) Mechanical obstacles to union.
(3) The absence of a sufficiency of bone tissue at the seat of fracture.

The first group may be subdivided into causes of local and causes of general failure of nutrition: of the local causes the most important is a lesion of the nutrient artery of the bone; of the general, all debilitated and cachectic conditions.

Under mechanical obstacles to union are included wide separation of fragments, insufficient immobilisation of frag-

ments and separation of the bone ends by the interposition of muscle, fascia, or aponeurosis.

An insufficiency of the essential bone cells that alone are the elements capable of giving rise to the new bone required for union occurs in many cases of extensive local bone destruction by trauma, infection, or new growth.

Such different causes give rise to three pathological types of non-union in simple fractures:

(*a*) The condition of absence of all attempt at repair occurs in cases of local bone disease and general debility. The bone ends remain attenuated and rarefied and united by a small amount of scar tissue.

(*b*) The commoner condition of fibrous union occurs in cases where an attempt at union on the part of the bone ends has resulted in the production, not of bone scar tissue, but of the ordinary *un*differentiated scar tissue of soft parts. The most common cause of this is delay in union between the two masses of bone granulation tissue or pre-callus either because of the interposition of some obstacle or from too wide a separation of the bone ends; too great a gap is to be bridged over. Where this is so the earliest formed granulation tissue has passed from its vascular stage of callus to its relatively avascular bone scar stage. Once this stage is reached in and immediately around the original bone ends, further growth of granulation tissue or callus in the direction of bridging the gap is not to be looked for. Further, the chance of its ever occurring rapidly diminishes with the lapse of time and with the progressive increase in density of the already formed bone scar tissue. This is absolutely analogous to the failure of replacement fibrosis of a large hæmatoma, except that the blood-clot that is unreplaced by osteoblasts comes

to be replaced by fibroblasts, so that a mass of ordinary scar tissue forms between and connecting the two masses of bone scar.

(c) The condition of true false joint or pseudarthrosis is merely a variety of this fibrous union. It is the result of constant movement taking place at the seat of non-union. In this way the sclerotic bone ends become smooth and eburnated, within the mass of scar tissue appear artificial bursæ or collections of fluid, the remaining tissue forming cords of ligamentous bands that unite the bone ends; so that all the elements of a joint are reproduced.

Failure of Spontaneous Completion of Repair in the Presence of Infection—in Infective Osteomyelitis.—Here, again, each of the causes of such failure bears a strict analogy to a corresponding cause of such failure in soft parts.

(1) **Failure of Repair due to the Presence of an Infected Foreign Body or Sequestrum that the Organism is incapable of absorbing or discharging.**—As long as a single sequestrum remains in the tissues, the occurrence of complete and final healing is out of the question. We have already seen how unreliable is the spontaneous discharge of sequestra in general, how next to impossible their elimination when encased. In many cases of chronic osteomyelitis, on their account alone spontaneous healing is not to be expected.

In the absence of a definite sequestrum, however, and especially in cancellous bone, healing fails because in pockets and recesses a balance has been struck between the organisms and the local resisting power, on the whole neither being able to make much, if any, progress against the other. In other cases the organisms have a very slight constant ascendancy over the local resistance, so that a very slow, gradual, but progressive process of bone destruction is taking place; under such circumstances spontaneous final healing is of course impossible.

(2) **Failure of Healing of an "open" Bone Cavity is Analogous in all Respects of its Pathogeny to Failure of Healing**

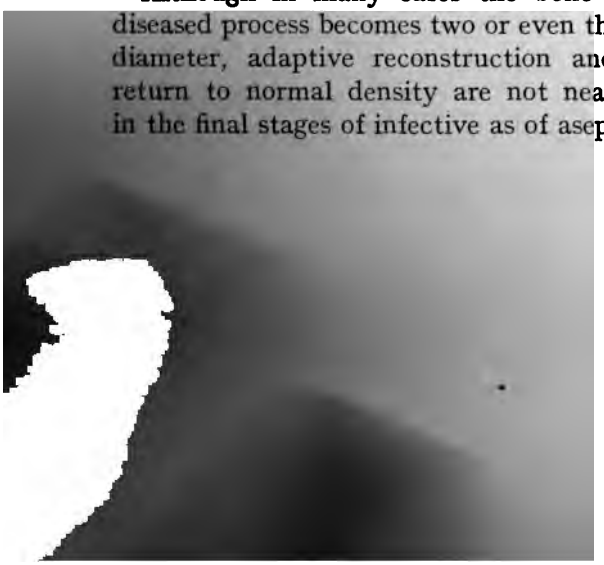
by Granulation.—In all such cavities that are even moderately large a chronic stage is reached, in which conversion of the earliest formed bone granulations in the floor and walls of the cavity into densely sclerotic bone results in the cutting off of an adequate blood-supply to the more lately formed osteoblastic cells, with consequent inhibition of their further proliferation. At the same time, and for the same cause, cessation of all epithelial ingrowth occurs.

(3) **Failure of Healing of a Closed Bone Cavity.**—What has been said in connection with the impossibility of spontaneous healing in such a cavity with rigid walls as that of chronic pyopneumothorax on page 42 holds in every particular as regards the healing of infected bone cavities. Such cavities have rigid walls incapable of collapse; hence healing by spontaneous approximation, sudden or gradual, of these walls becomes a physical impossibility. The only possible way in which healing by secondary adhesion could take place would be by so exuberant a growth of granulations from the walls as would rapidly cause the obliteration of the cavity. In osteomyelitic cavities, however, especially where of traumatic origin, the stage when this is possible has already come to an end by the time that union has been effected, and is usually long past by the time the sequestra come to be removed. The reparative process, up to a certain point, has proceeded satisfactorily; it has not been long, however, before the walls have become, from development and persistence of the osteosclerotic process, progressively denser and thicker and less vascular, thus putting a premature and effective check on cell proliferation and the formation of further granulation tissue. This series of pathological events it is that explains the cause of failure of healing of cavities and tunnels in bone long after all sequestra have been removed, all foci of carious bone excavated—long after all actual bone disease is at an end. Healing in such cases is delayed, not because of the presence of sepsis—frequently on culture the discharge will be found to remain sterile over long periods; but because of the mechanical impossibility of the collapse of its walls. As

long as the cavity remains patent and wholly unobliterated so long will the sinus remain open. Antiseptic and physical agents directed against the infection will be found unavailing; operation alone, and only an operation that is sufficiently radical and ensures the elimination of the cavity, is able to assure completeness and finality of healing.

There is a further point in the healing of infected bone cavities worthy of notice; not only is the repair so often incomplete in the sense of complete healing being indefinitely postponed, but also in the sense that such repair as does take place is so often atypical—bone loss is not replaced wholly by bone scar tissue. Even where a small cavity has completely healed by secondary adhesion of its walls, skiagraphy shows that the central mass of new tissue is not, and does not, become osteosclerotic, but merely ordinary scar tissue. A similar phenomenon is seen clinically in cavities that have almost completely healed. Although they have enormously thickened bone walls, yet inside these a considerable thickness of ordinary scar tissue seems constantly to persist. This seems to indicate that the later final stages of bone repair seem to be far more sensitive to a decrease in blood-supply than the earlier; at any rate in the absence of an adequate supply, they do not proceed so far as the earlier.

Although in many cases the bone at the seat of the diseased process becomes two or even three times its normal diameter, adaptive reconstruction and the tendency to return to normal density are not nearly so well marked in the final stages of infective as of aseptic repair of bone.



CHAPTER III

THE PATHOLOGY OF COMPOUND FRACTURES AND OF CHRONIC TRAUMATIC OSTEOMYELITIS

THE following anatomical types of compound fractures represent not only such various pathological conditions, but also such different therapeutic problems as to justify their separate description and consideration:

A. Compound fractures in which infection is absent from the beginning or else fails to develop.

B. Infected fractures that develop into cases of osteomyelitis.

(1) Complete fractures of the shafts of long bones with or without displacement, with or without comminution.

(2) Incomplete fractures.

(*a*) Of the shaft of long bones—in which the whole circumference of the compact cylinder is not fractured through, and in which the infection does or does not gain access to the medullary canal.

(*b*) Of short bones and of epiphyses, in which the infection gains access to a mass of cancellous tissue.

(*c*) Of flat bones, in which a layer of cancellous tissue lies enclosed between two plates of compact bone.

C. Infected articular fractures that become cases of septic arthritis as well as cases of osteomyelitis.

A. Compound Fractures in which Infection is either Absent from the Beginning or else fails to Develop.

Pathologically such cases among gunshot fractures may be divided into three classes :

(1) Cases of compound fractures, complete or incomplete, with more or less comminution, with or without retention of the projectile, but with complete absence of all signs of infection.

In the great majority of these the projectile is a rifle or machine-gun bullet, less often a shrapnel ball. Practically all cases of fracture due to shell or bomb fragments suppurate unless a radical operation has been performed early. In many ways such fractures of this first class resemble both clinically and pathologically simple fractures, and up to a point may be treated as such ; but one must not too early conclude that infection is absent, often it is merely latent ; its secondary development " occurs especially, though not inevitably, when foreign bodies remain in or near the bone, and especially often also in wounds due to bullets whose covering has been torn or that have broken up into fragments."

Where the projectile has not been retained in the seat of fracture union and repair take place through a series of pathological changes indistinguishable from that obtaining in simple fractures. In many such, however, as in a greater proportion of cases in which one or more pieces of the projectile have been retained the infection, though often localised, remains latent and liable to develop into activity, insidiously, and on some later occasion. Where a foreign body becomes embedded in the mass of callus it acts as an aseptic irritant, keeps up the vascular, and defers the dense sclerotic stage of the bone scar, and so delays the attainment of functionally useful union. The bone at the seat of union often remains hot and tender for a long time.

(2) The second class of case is that in which, although the skin is broken, the projectile has never actu-

ally come into contact with the bone. Although infection has occurred in the soft parts, the seat of the fracture, which is really a subcutaneous fracture, remains shut off from contamination by the infection.

(3) The third variety consists of those cases—most commonly of incomplete fractures—referred to in the Introduction to this book in which the prophylaxis of osteomyelitis has been successfully effected—such cases as those reported by Carrel, Leriche, and Bowlby.

In these aseptic repair and union obtain, although it is to be expected that in a certain number, the infection being merely latent, the micro-organisms will after months of inactivity declare their presence by the onset of acute or chronic osteomyelitis.

B. Infected Fractures that develop into Cases of Osteomyelitis

(1) **Complete Fractures of the Shafts of the Long Bones.**—Variations in two factors, the amount and character of the displacement, and the extent of the bone loss determine the different pathological types presented by cases of complete infected fractures of the shafts of the long bones.

What we have written regarding the effects of displacement of fragments on the shape of the fracture cavity, and on the peculiarities, difficulties, and occasional failure of union in simple diaphyseal fractures, applies with equal if not greater force in the case of infected fractures.

The total ultimate bone loss in such cases is represented by the sum of the loss due to the actual violence with that due to the original and any later stage of acute infection, in other words by the primary, secondary, and tertiary sequestra in addition to any bone that may have been pulverised or completely shot away, as well as such as has been destroyed by caries. The bone thus actually lost,

LANE LIBRARY

however, represents as well a corresponding loss of a potential source of the new bone required in the process of repair. Regarded in this light it is as it were lost twice over. Hence a study of the sources and modes of formation of new bone in such fractures becomes a matter of the first importance; this at once raises the question of the periosteal origin of such bone, and the answer to this question will of course supplement the evidence supplied by experimental and human surgery concerning the osteogenetic powers of the periosteum.

By his experiments Macewen may justly claim to have established the fact that the periosteum is essentially a limiting membrane, setting a peripheral limit to bone growth, and that it derives whatever osteogenetic powers it possesses from the proliferative and formative activity of the bone cells or osteoblasts, that, having been detached with it, adhere to and are nourished by it. "If the term periosteum is applied, as it ought to be, simply to the fibro-elastic layer, then it may be held that this is nothing more than a limiting membrane, without any osteogenetic power. But if by periosteum is meant the fibrous layer with the bone cells adhering to its deep surface, then it has osteogenetic power, just in proportion as it takes with it this part of the living bone." This quotation may be taken as stating the position fairly; the practical question remains, however, and it has not been answered by any series of experiments—In what proportion of cases of simple fractures do such osteoblasts become detached with the periosteum and come to play an important part in the formation of the external callus? It seems to the writer that the numerous severe compound fractures of the present day furnish abundant and valuable evidence in favour of the view that such osteoblastic detachment not only is practically universal in fractures, but also plays an important rôle in the production of union.

In so many infected gunshot fractures bony union takes place alone by external or subperiosteal callus formation. What is its origin? Let us take for example the case, typical

of thousands of such cases, represented by the skiagram that forms the frontispiece of this book. A gunshot fracture of the femoral shaft has resulted in the comminution and necrosis of 1½ to 2 ins. of the whole circumference of the shaft. A casing of new bone has been formed round the sequestra, and from this a closed bone cavity has resulted, whose walls consist of dense bone-scar tissue ; the upper and lower limits of this cavity represent the densely sclerosed bone ends left after separation of the necrosed bone, the opening of each end of the medullary canal having been closed by a plug of " internal " bone scar, whilst the peripheral walls are formed by the " external " or subperiosteal bone scar or involucrum. Inside the cavity exist the remains of the original shaft of the bone in the shape of sequestra that, on removal, could be so pieced together as to re-form the original outline of this portion of the shaft. What, then, is the origin of the dense thick cylinder of involucrum ?

Its formation cannot be attributed to what Macewen attributes it in spontaneous osteomyelitis—an outpouring of osteoblasts into the areolar layer of the periosteum before its detachment takes places, the initial result of the infective irritation of the bone tissue ; for in these cases of traumatic osteomyelitis the periosteal detachment precedes by at least some hours the stimulating action of the infection on osteoblastic proliferation. Again, the growth of this involucrum is of so uniform a thickness and density that it can hardly arise from the osteoblasts of such incompletely detached fragments as survive the infection ; the occurrence of such fragments, moreover, is by no means invariable. A similar formation of involucrum takes place in the numerous cases reported by Leriche in which immediate subperiosteal removal of all fragments, loose and attached alike, has been carried out before the onset of the infective activity.

On the other hand, it can hardly be the result of a mere extension of osteoblastic growth from the ends of the shaft, for it has often come into existence before the process of sequestration is complete ; it is also exceedingly unlikely that growth should take place under such circumstances

across such a gap as exists between the bone ends when in Macewen's experiments bone tissue would not grow under aseptic conditions across a gap of 1¾ ins. If, as in the radical operation for obliteration of such closed cavities, one removes subperiosteally one half of the involucral cylinder besides all the sequestra, re-formation of further subperiosteal bone takes place with extraordinary rapidity, so that in a few weeks the bone is as thick as ever. Such growth can hardly have resulted from the inner aspect of the densely sclerotic avascular remaining walls of the cavity; it can have taken place only by active proliferation of bone cells adherent to and detached with the periosteum. Further evidence in favour of the periosteal origin of the involucrum is furnished by a study of the cases where union fails to take place and of the cases in which union occurs between only one half of the compact cylinder of the shaft. The great majority of cases in which Leriche performed immediate subperiosteal removal of all fragments showed that, no matter how great the distance between the bone ends, as long as the periosteum is left more or less intact so long will non-union be avoided. On the other hand, non-union seems to occur in gunshot-fractures only in cases where extensive bone loss is accompanied by extensive periosteal destruction. Where complete loss of the whole circumference of a segment of the shaft with one half of the circumference of the periosteal sheath has taken place, there results but a semi-cylindrical involucrum and an open bone cavity.

It seems to the writer, then, impossible to resist the conclusion that the occurrence of adhesion of bone cells to the detached periosteum is practically universal in fractures both simple and infected, and that by reason of these the periosteum, besides acting as a limiting membrane, plays a part in the union—a part in simple fractures of secondary, in compound of supreme importance. It is not indeed a parent or generator of osteoblasts and bone tissue, but protects them from the infection and subsequently provides them with nutrition as, at any rate in compound fractures, the chief factor in their growth and development into the

subperiosteal bone scar or involucrum that alone in so many infected gunshot fractures assures the union.

The different results in the direction of periosteal osteogenesis observed by different surgeons after subperiosteal operation would appear to be largely due to different methods and instruments used in detaching the periosteum. Leriche, following Ollier, uses a razor-edged rugine, and claims the constant retention of osteogenetic power by the periosteum, whilst Macewen probably got negative results in animals because of the great care he took not to encroach on bone tissue proper; in one case, indeed, of heterotopic periosteal transplantation, he did get new bone formation and explained it in the following manner: "Doubtless this nodule resulted from a minute osseous plaque, or from some osteoblasts adherent to the periosteum in the process of removal."

On these three factors, then—the amount of periosteal destruction as well as the extent of the actual bone loss and the degree of displacement—will depend the whole aspect of the pathological condition and the therapeutic problem. These determine not only the amount and rapidity of the union, but the size, shape, and contents of the sequestral cavity. Before commencing the treatment of any case, the surgeon should, after thorough clinical and skiagraphic examination, consider the case in terms of these three factors, and on this plan and with the evidence thus obtained as material, build up in his imagination an accurate picture and realisation of the full details of the actual pathological condition present. It is important, then, that we should study in turn the most common general types of this form of chronic traumatic osteomyelitis.

TYPE I.—A transverse fracture of the shaft with little or no comminution, little or no displacement.

Regarded from the pathological and therefore from the therapeutic standpoint, this is the simplest type of all. *Vide* Diagram 6 and Plates I, III, and IV. In such a case loss of bone tissue is wholly the result of the infection, not

at all the direct result of the original violence. Its extent depends entirely on the severity of the supervening infection, of the inflammatory reaction. In many cases such destruction is but slight. A small, irregular piece of each fractured end undergoes necrosis, and in the course of time becomes separated to form a secondary sequestrum; of primary sequestra there are none.

No displacement having taken place, the three zones of

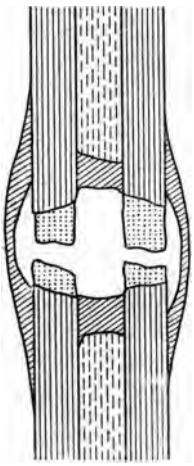


DIAGRAM 6.—The sequestral cavity of an infected transverse fracture without comminution or displacement; only secondary sequestra in the process of formation; the bone united by subperiosteal new bone only.

the fracture cavity are present from the beginning: the outer due to detachment of periosteum, the intermediate, destined to become subsequently enlarged by the separation of the necrosed bone, the internal, variable in extent with the amount of destruction of the marrow tissue in the medullary canal.

The periosteum that has been suddenly stripped off the cylinder of the shaft by the violence of the original trauma has taken with it a layer of bone cells that by its blood-supply are enabled to survive the infection; henceforward

PLATE I. CASE 1.—Chronic osteomyelitis of the shaft of the fibula with an antero-posterior tunnel containing several very small secondary sequestra.

PLATE II. CASE 1.—Showing the result of the radical operation; removal of the sequestra with subperiosteal resection of the outer wall of the tunnel.

PLATE III. CASE 2.—Chronic osteomyelitis of the shaft of the fibula with a large antero-posterior tunnel. The sequestra have all been previously removed without healing resulting.

PLATE IV. CASE 3.—Chronic osteomyelitis of the shaft of the humerus, showing a large cavity lying between anterior and posterior walls.

the periosteum is an osteogenetic as well as a limiting membrane. Where the projectile has passed completely through the bone, the periosteal sheath will show at least two rents with a variable loss of substance; where the foreign body is retained in the seat of fracture very often but one. Through these rents the fracture cavity—and subsequently the sequestral cavity—communicates with the wound of the soft tissues and the exterior. These subsequently develop into the cloacæ.

With the subsidence of the acute stage of the infection and inflammation during which the original blood-clot of the cavity becomes converted into pus, and a variable amount of bone tissue necroses, reparative phenomena make their appearance; the process of sequestration, with its inevitable destruction of a zone of living bone tissue, and with its rarefaction of a further zone, leads on to the formation and outgrowth of an osteoblastic granulation tissue. This grows in all directions, outwards to help fill the contiguous part of the external zone, downwards to meet that sprouting from the opposite bone end—though here its extension is soon prevented by the ever-present acute irritation of the sequestra—inwards across the medullary canal, which thus becomes sealed off.

Such granulation tissue becomes callus, in time densely sclerotic bone tissue; hence before many weeks have passed the new bone ends have become harder and denser and less vascular than even normal compact bone tissue, and the medullary canal shut off by a plug of similarly sclerotic bone; further growth of granulation tissue from bone scar thus becomes very slow and ineffective, if not impossible. Where but a slight amount of bone tissue has been lost, and the gap between the bone ends is quite narrow, union across it is frequently accomplished. In many other cases where the gap is at all wide, the process of bone regeneration from the main fragments comes to a definite end before such union across intermediate and internal zones of the fracture cavity can take place, and a permanent cavity containing the sequestra and lined by weak, ill-nourished granulation

tissue remains. In such a case the sole agent of bony union is the involucrum—a cylinder of new bone laid down in the outer zone by the activity of the periosteal osteoblasts.

Such gaps in the periosteum as we have referred to persist as gaps through this involucrum and form cloacæ, with edges formed of granulation tissue the further growth and development of which into bone with obliteration of the passage is usually sufficiently well prevented by the constant irritation of the discharge that flows through them from the sequestral cavity.

The sequestral cavity in such cases, simple and regular in shape, variable in size, and containing a certain number of sequestra, represents a pathological condition that, except for progressive increase in the thickness and density of its walls, is absolutely stationary, and that, on account not only of its sequestra, but still more of the dense sclerotic nature of those walls, remains quite incapable of spontaneous obliteration and healing.

An exacerbation of intensity of the infection and consequently of the inflammatory reaction in such a cavity will be extremely likely to result in further extensive necrosis of the dense bone scar of its walls.

TYPE 2.—A transverse fracture of the shaft with little or no comminution, but with considerable displacement. *Vide* Diagram 7 and Plates V. and VI.

In such cases the bone loss is largely if not wholly the result of the infection, and is represented by the secondary sequestra. The degree of displacement, especially where there is overlapping of the ends of the bone, results in a very complicated fracture cavity, the three zones being practically merged into one large subperiosteal zone. The detached and now osteogenetic periosteum shows a variable number of gaps or rents destined to form cloacæ. Sequestration, accompanied by reparative phenomena, takes place; the ends of the medullary canal usually become shut off; the bone ends, at first rarefied and later dense and sclerotic, take but little part in the subsequent union, although in

PLATE V. CASE 4.—Chronic osteomyelitis of the shaft of the femur; formation of a tunnel by deposit of new bone around a drainage tube.

PLATE VI. Case 6.—Chronic osteomyelitis of the shaft of the femur; there has been little comminution but great displacement of the fragments; no formation of a real sequestral cavity; a few secondary sequestra and many inserted pieces of metal embedded in all parts of the callus.

cases of overlapping fusion takes place over the slight extent of their outer surfaces that are in contact. Union is almost wholly the result of subperiosteal osteogenesis.

The shape and size of the sequestral cavity that results

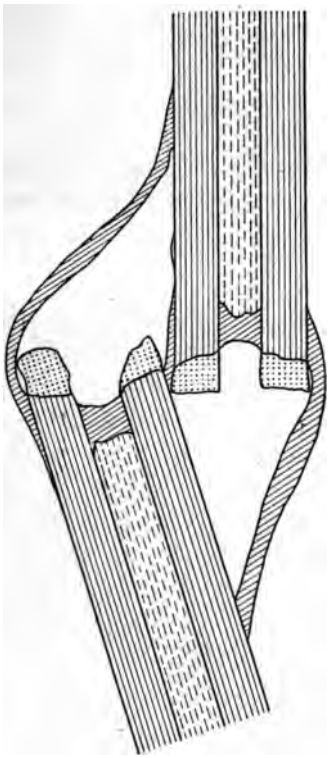


DIAGRAM 7.—The sequestral cavity of a transverse fracture without comminution but with displacement; secondary sequestra only, the bone united chiefly by subperiosteal new bone but also slightly by direct union across the surfaces of the fragments that are in contact.

depends entirely on the amount of the bone loss; where necrosis has involved a considerable extent of either end of the shaft, the cavity is large and single, forming an oblique cylinder; where the amount of necrosis has been slight

and the bone ends have remained in some sort of lateral contact, the cavity, whilst actually more complicated, becomes really divided into two smaller compartments, thus resulting in no such large "dead" space the obliteration of which presents the chief difficulty in treatment. Indeed, in cases where but slight necrosis has followed slight overlapping these two compartments are so small that, after the simple removal of sequestra, they often undergo spontaneous obliteration.

TYPE 3.—A transverse fracture of the shaft with considerable comminution, but without much displacement of the chief fragments. *Vide* Diagram 8 and Plates VII., VIII., and IX.

The total bone loss in such cases is always considerable and represented in part by primary sequestra, in part by secondary; the displacement is slight, but usually the three zones become largely indistinguishable, because of the irregular distribution of the bone fragments throughout the fracture cavity; the periosteum in these cases usually suffers a considerable amount of laceration. The reparative process comes to the usual indeterminate end; the involucrum, enclosing peripherally a very large but fairly regular sequestral cavity, forms the sole effective element in the union of the bone.

TYPE 4.—A transverse fracture with considerable comminution, and a considerable displacement. *Vide* Diagram 9, Plate X.

In such cases the general condition is the same as in a fracture of Type 2, in which the bone loss has been great, and the resultant union wholly due to the involucrum. The cavity is large, irregular, and filled with numerous primary and secondary sequestra. The bone at the seat of union in such a case is frequently three times its normal diameter.

Most comminuted fractures of the shaft are included in cases of transverse fracture; most cases of a distinctly oblique fracture are cases with little or no comminution.

PLATE VII. CASE 7.—Chronic osteomyelitis of the shaft of the fibula an antero-posterior tunnel containing a small sequestrum

PLATE IX.—Chronic osteomyelitis of the shaft of the humerus; a large tunnel with very thick walls.

PLATE X. CASE 8.—The end result of a case of chronic osteomyelitis of the shaft of the femur; numerous sequestra were removed; subperiosteal resection of the outer wall of an enormous antero-posterior tunnel; so much bone was removed that fracture of the shaft occurred and rendered possible correction of the deformity. This plate was taken after healing and sound union.

PLATE XI.—Chronic osteomyelitis of the shaft of the femur; an oblique tunnel with several sequestra.

TYPE 5.—An oblique fracture of the shaft without displacement. *Vide* Diagram 10 and Plate XI.

In such cases usually very little bone is lost; usually merely a thin layer of bone necroses at either end to form secondary sequestra; primary sequestra are usually absent.

DIAGRAM 8.—The sequestral cavity of a transverse fracture with comminution but without displacement; primary and secondary sequestra, union by subperiosteal new bone only.

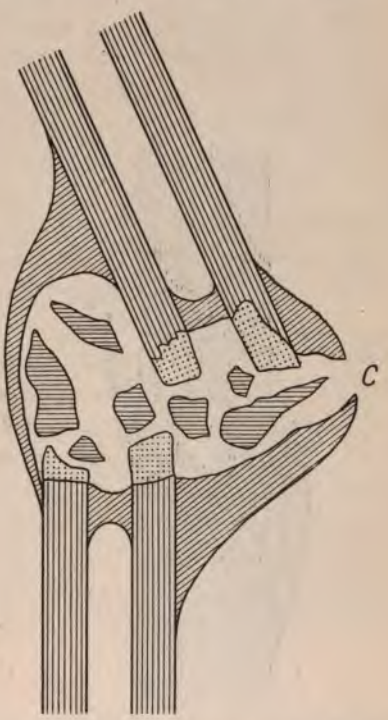


DIAGRAM 9.—The sequestral cavity of a transverse fracture with comminution and displacement; primary and secondary sequestra; union by subperiosteal bone only; cloaca shown at C.

It is rare in such cases for a slight amount of lateral displacement not to take place; but, where this is slight, the fracture cavity consists of a narrow oblique cleft. The periosteum has usually been torn, at any rate at two spots,

in some cases, however, at one only. By the time sequestration is complete union has usually been effected, largely by subperiosteal bone formation. Where the amount of bone loss has been very slight further union may take place

DIAGRAM 10.—The sequestral cavity of an oblique fracture without comminution or displacement; secondary sequestra only; union by subperiosteal bone only; Cloaca shown at C.

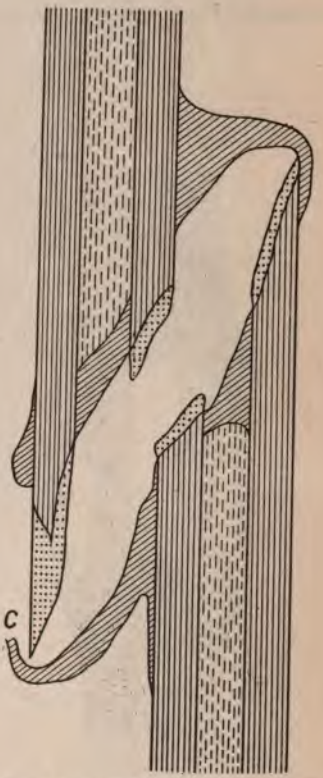


DIAGRAM 11.—The sequestral cavity of an oblique fracture without comminution but with displacement; secondary sequestra only; union by subperiosteal new bone; cloaca shown at C.

subsequent to simple sequestrectomy by means of the internal and intermediate callus as well; where, however, this does not occur the sequestral cavity remains indefinitely, long and narrow, lined with granulations, and opening by one or more cloacæ into the soft parts.

TYPE 6.—An oblique fracture with considerable displacement. *Vide* Diagram 11.

It is among cases of this type that sequestral cavities of the greatest size usually occur; this is not at all because of extensive bone destruction, but because of the wide separation of the very extensive oblique fractured surfaces. In point of fact, the absolute bone loss is usually but slight; the original trauma causes but little fragmentation; the infection usually results in long but narrow secondary sequestra that represent chiefly the extreme pointed ends of the bone fragments. The displacement is usually that of lateral separation of the fragments with overlapping, and the greater this lateral displacement and overlapping the wider the fracture cavity and the greater its subperiosteal zone. The occurrence of sequestration leads to a further increase in the width of the sequestral cavity.

In these six types of infected diaphyseal fractures, neither the original injury nor the subsequent infection has resulted in more than local spots of laceration and destruction of the periosteum. Extensive periosteal destruction occurs, however, especially often, as might be expected, in cases of extensive comminution. This, which in simple fractures would not perhaps be of any very great practical significance, becomes in infected cases of very great importance. There are two general types of such cases, with extensive periosteal destruction.

TYPE 7.—Comminution of a whole segment of shaft with destruction of a considerable part of the corresponding periosteal sheath. *Vide* Diagram 12.

In cases in which a definite segment of the periosteum has been destroyed over one half of its circumference, together with complete comminution of the whole circumference of the corresponding bone segment, new bone is laid down beneath the intact portion of the periosteum only; union takes place through this semi-cylinder of

involucrum and through this alone. The resulting sequestral cavity is an open one, from which the spontaneous emergence of sequestra becomes unusually easy and their surgical removal a very simple operation.

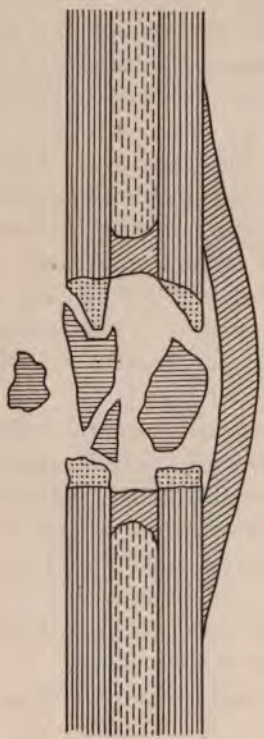


DIAGRAM 12.—The open sequestral cavity of an infected fracture with complete comminution of a segment of the bone, destruction of one half of the circumference of the periosteal sheath, primary and secondary sequestral union by a semi-cylinder of subperiosteal involucrum.

DIAGRAM 13.—Failure of union with absence of sequestral cavity; no involucral sheath consequent on the destruction of the periosteal sheath as well as of a complete segment of bone; primary and secondary sequestra.

Where the periosteum survives not in one single sector, but merely in strips and shreds, the attainment of functionally useful union will be long delayed, although in most cases eventually assured.

TYPE 8.—Comminution of a whole segment of the shaft with destruction of the whole corresponding segment of periosteum. *Vide* Diagram 3.

In such cases one of two things happens: either union fails altogether, or else it takes place at the expense of shortening of the limb by an elevation of the lower fragment into contact with the other. The former course is almost invariable in the case of the humerus or of one of such a pair of bones as the radius and ulna; the latter occurs commonly in the femur. In the latter case the strength of contraction of the muscles attached to the lower bone fragment is responsible for so favourable a consummation; non-union in compound fractures of the femoral shaft very rarely occurs.

Such is a more or less schematic description of the general types of these cases; all the possible variations in such details of the pathological process as the size, shape, number, and nature of the sequestra, the size, shape, contents of the sequestral cavity, the dimensions and relations of the involucrum, in individual cases result in no two cases being alike even of those of the same general type or class. Many cases combine characteristics of two or more of these types; others are so complicated, so atypical as to defy classification into any of these categories.

It is not uncommon to meet cases in which an uncomplicated fracture of one half of the circumference of the shaft coexists with an oblique or comminuted fracture of the other half, often with great displacements of its fragments. In certain cases one or other end of the medullary canal fails to become occluded; an important variety of this is due to the original lodgment of a large splinter of bone within the medullary canal. As a primary sequestrum this effectually prevents its closure by new bone formation, and so converts this end of the medullary canal into a part of the sequestral cavity.

(2). (*a*) **Incomplete Fractures of the Shafts of Long Bones.**—These vary from a slight gutter wound to an almost

complete fracture of the circumference of the shaft. What is pathologically important in each case, however, is the question of the involvement or non-involvement of the medullary canal in the infective process.

Cases where the Medullary Canal has not been opened up to the Infection.—The usual phenomena of comminution, necrosis, and new formation of bone occur in these cases as in others; and with the same results. Where comminution has taken place primary sequestra will be present, but these will often be found to lie free in the soft tissues, and but rarely to be encased within a sequestral cavity. The opposite is the case, however, with such secondary sequestra as result from the almost inevitable necrosis of a greater or less extent and thickness of the compact bone of the shaft; only too often in cases where the periosteum has escaped destruction, before the process of sequestration is complete, an involucrum has come into existence to form a future barrier to the spontaneous emergence of the sequestra; thus we have the by this time familiar condition—sequestra imprisoned within a sequestral cavity the walls of which undergo with the passage of time a progressive sclerosis and increase in thickness, so that even after the removal of the sequestra a cavity remains only too frequently incapable of spontaneous obliteration. Occasionally the accumulation of new bone, dense and sclerotic, around a small sequestrum or cavity doubles the normal circumference of the bone.

Cases where the Medullary Canal is opened up to Infection.—In such cases very often quite an extensive amount of comminution and necrosis occurs. By the time the chronic stage is reached, however, the pathological condition of things is more or less closely identical with that described above under Types 3 and 7, and the sequestral cavity that results may be either an open or a closed one.

(2). (*b*) **Infected Fractures of the Short Bones and of the Epiphyses.**—Masses of cancellous tissue—the characteristic structural feature of such bones—are of course favourite [illegible]or the development of spontaneous osteomyelitis,

especially of the osteomyelitis, subacute or chronic, that occurs in the non-pyogenic tubercular infections. Their involvement in an infective osteomyelitis of traumatic origin is, however, but rarely met with in civil life, nearly all the ordinary compound fractures of industrial and street accidents being diaphyseal. On the other hand, infected gunshot fractures of the epiphyses and of the tarsal bones, even when considered relatively to the number of gunshot fractures of the shafts of long bones, have proved especially common in this war.

The open cancellous structure of the infected bone is the cause of the development of special pathological features in these cases, whilst the proximity to joint surfaces of the osteomyelitic focus is of special importance and significance in the treatment of the acute and chronic phases alike.

The characteristic pathological feature of compound fractures of these bones is their cavitation. The outer shell of the bone offers no such resistance to the passage of the projectile as does the thick compact cylinder of the shaft of long bones ; the occurrence, therefore, of extensive splintering and comminution is uncommon ; the damage inflicted on the bone is much more often local, merely a direct penetration or perforation of the spongy mass. For the same reason any metal that has penetrated, but, having failed to perforate, is retained in the bone, is almost always a fragment of shell or bomb, occasionally a shrapnel ball, practically never a rifle or machine-gun bullet. These last, except when nearly spent, always possess more than sufficient momentum to force their way through the whole thickness of the bone. For a like reason, shrapnel balls, when embedded, will rarely be found broken or deformed. The immediate result, then, of this penetration of the bone is the production of a tunnel or cavity which passes either completely or partially through the substance of the cancellous tissue ; in the latter case the infected foreign body is retained and occupies part of the cavity.

It is in the neighbourhood of this foreign body that the infection develops in its greatest intensity and the resulting

pathological changes in the bone become most marked; whilst that part of the bone relating to the rest of the cavity undergoes changes that are identical in character though they do not usually occur to so great a depth. The development of this infection is in most cases rapid. Its direct destructive action is soon added to that of the original trauma; small fragments and splinters of bone already isolated by the violence of the missile soon succumb to form **primary sequestra.** The bruised, irregular walls of the cavity, especially where in contact with the source and origin of the infection, are attacked; the cells of the bone marrow, lying open to the infection between the bony trabeculæ, undergo necrosis; the vital essential bone cells die, whilst the matrix of bone earths becomes eroded and disappears. In other words, acute ulceration or caries of the bone sets in over the whole extent of the walls of the cavity and to a depth that varies with the virulence of the infection and the powers of resistance, local and general, of the patient. In places here and there small scales of bone and pieces of trabeculæ may undergo an actual necrosis and either be quickly set free by the rapid advance of the carious process beyond them or remain unseparated until such time as loosened by the gradual subsequent process of bone ulceration. Such become **secondary sequestra,** respectively early and late. Necrosis, however, plays but an occasional and then subsidiary and unimportant part; the principal process in this extension of the limits of the original cavity is that of caries. Cancellous bone has too efficient a blood-supply, and is of too open a structure to undergo massive necrosis on any but the smallest scale.

Tissue reactions in defence soon make their appearance, clinically by swelling of the soft parts with an abundant serous discharge, pathologically by the vascular changes followed by exudation, phagocytosis, and bacteriolysis that bring about the commencement of suppuration, and the serous discharge is replaced by pus. In cases where adequate drainage has either occurred naturally or been provided by surgical intervention, the early lining of the cavity with

granulation tissue marks the commencement of the reparative process.

The destructive effects of the infection, however, are not always restricted to the bone itself. Epiphyses and such short bones as those of the tarsus lie in intimate relation to one or more joint cavities. Primary joint involvement, then, may occur:

(*a*) Where the projectile has traversed part of the joint cavity as well as the mass of cancellous bone. Especially often does this occur where an epiphysis, as in the hip joint, lies wholly or largely within the articular cavity.

(*b*) Where the missile, without actually itself penetrating the joint cavity, has nevertheless opened it up to microbic invasion by splintering and comminution of the bone.

(*c*) Where the thin layer of bone lying between the infected cavity and the joint becomes eaten away during the acute stage of the destructive action of the bacteria.

The original process of bone destruction may, however, reach and involve surfaces other than the articular; the periosteum over such a surface becomes congested and thickened, and then raised from the bone by the formation of an abscess, which, eventually bursting or being incised, develops into a chronic sinus; through such sequestra, or even the foreign body itself, may ultimately be discharged. Later extension of similar bone destruction and cavitation will take place, moreover, whenever blocking of the exit of the whole or of part of the osteomyelitic cavity takes place. Sudden impaction of the foreign body or of sequestra within the mouth of the sinus, its more gradual occlusion by progressive contraction of the scar tissue in its walls, inadequacy of drainage from whatever cause has this one inevitable result; the subsequent retention of the pus immediately intensifies the destructive activity of the germs, and further

progress in different directions of the carious process results in corresponding increases in the size of the cavity, often in fresh abscess and sinus formation, and not infrequently in secondary or late involvement of articular surfaces and of joint cavities.

Destructive changes in joints leading subsequently to various forms and degrees of ankylosis and stiffness are often due, however, not to the involvement of the joint tissues in the infective process itself, but merely to their falling within the range of the primary or secondary inflammatory reaction. An arthritis by contiguity of inflammation, and not by actual communication of infection, is thus set up. In the later more chronic stages of ankylosis and stiffness, however, it is often impossible to decide whether the articular lesion should be attributed to a more than usually severe aseptic inflammation by contiguity or to a less than ordinarily mild attack of septic arthritis by direct communication.

By the time the walls of the cavity are lined with granulations the progress of the destructive action of the infection has come to an end; by this time bacteriolysis and phagocystosis have long since gained a relative mastery over the micro-organisms, and this, with slight fluctuations, they usually retain so long as drainage remains adequate, and surgical interference with the infected focus be abstained from; henceforward the discharge lessens in amount, contains both fewer varieties and fewer absolute numbers of germs and often for long periods is rather serous than purulent. During this time the tissues are engaged in making an effort at repair of the damage and destruction. Even in the absence of all foreign bodies and sequestra, however, and despite the fact that thickening of their walls by osteosclerosis is not at all a marked characteristic, and despite also the adequacy of their blood-supply, the spontaneous repair of such infected cavities only too often falls short of completion. Even with complete obliteration by spontaneous granulation of a cavity in such bones, in very many cases destruction with subsequent replacement

by dense scar tissue of the skin and soft parts covering them would alone constitute sufficient cause of failure of complete and stable spontaneous healing of the wound. In such cases most frequently the sinus or chronic ulcer of the soft parts and the bone cavity itself together form a dead space incapable of spontaneous obliteration, either by granulation or by collapse and secondary adhesion of its too bloodless and rigid walls.

Such a condition, absolutely stationary as regards progress of repair over long periods of months, and even of years, is only too commonly met with in the condyles, great trochanter, and head and neck of the femur, in the upper end of the tibia and in the tarsal bones. This type of chronic osteomyelitis reaches its extreme example in cases of complete excavation of both femoral condyles and also in certain cases where a large tunnel with dense sclerotic walls exists, passing completely through the posterior part of the foot, involving the bones, and the soft parts on either side of them. To bring about final healing in such cases is one of the most difficult problems confronting the surgeon at the present time.

(2). (*c*) **Infected Fractures of the Flat Bones.**—These bones resemble the shafts of the long bones in the thickness and density of their walls, the short bones in their amount of cancellous tissue. Hence it can easily be deduced that, as regards the pathological phenomena they exhibit when the seat of traumatic osteomyelitis, they occupy a position midway between the shafts of the long bones on the one hand, and on the other the short bones and the epiphyses. On the whole, however, although in places in these flat bones, *e.g.* along the attachment of the scapular spine, and in the tuber ischii, cancellous bone is present in considerable quantity, they contain more compact and less cancellous tissue than the short bones; and contrariwise less compact and more cancellous tissue than the shafts of the long bones. Unlike these latter, they contain no medullary canal.

In gunshot fractures of these flat bones the mere mecha-

nical effects vary greatly not only with the nature and velocity of the projectile but also according as to whether a surface or a border of the bone receives its impact. Very often in the case of rifle and machine-gun bullets striking a surface tangentially their momentum is sufficient to drive them completely through the thin layer of bone—a mere perforation with little or no comminution resulting. Such fractures frequently remain aseptic. Others suppurate and become cases of acute and chronic osteomyelitis. In these latter the small plug of bone drilled out by the bullet, and more or less displaced, soon becomes a primary sequestrum; the edges of the tunnel formed by its displacement are attacked by the infection, and usually on a small scale, undergo necrosis, giving rise in time to numerous small secondary sequestra. A certain amount of destruction by caries of the cancellous bone lying between the compact plates also takes place. In such cases, after the performance of sequestrectomy, the periosteum or, where this has been destroyed, the soft tissue in relation to each surface of the bone, either falls together at once or is gradually drawn into contact by a cicatricial process with consequent obliteration of any such small cavity or tunnel as may have remained after the removal of dead bone. Hence the occurrence in these of sound spontaneous healing is not at all uncommon.

Shrapnel balls, and very occasionally fragments of shell and bomb, may act similarly; but, striking tangentially, these usually inflict far more serious damage, with extensive comminution of the bone; whilst they themselves either penetrate the bone completely or, failing this, are retained amid the bone splinters and fragments; although, where they have struck the bone surface at a very acute angle, they may be reflected back into the soft tissues. The factors in this direct trauma that are of chief pathological significance are in the first place the comminution, in the second the stripping up or partial destruction of the periosteum on one or both surfaces of the bone. The fragments are of all sizes, some quite free from all attachment, others

still adherent in part, at least to periosteum, and through this to other tissues.

The superimposition and development of an acute infection in such an extensive area of severe bone damage rapidly leads to the occurrence of necrosis of all the fragments that are quite loose or whose slight attachment to periosteum has not assured them a blood-supply sufficient to withstand the infection. Usually all fragments loose and adherent alike share this fate, the loose immediately becoming early primary sequestra, and the adherent, soon set free by ulceration through the tissue to which they have remained attached, forming later but still primary sequestra. Meanwhile, in most cases such corners, edges, and surfaces of the intact bone as lie directly exposed to the infection undergo necrosis to a depth variable in different cases, but usually bearing a direct relation to the extent of the denudation of their periosteum that has originally taken place.

To these destructive effects due to the infection, must be added in certain cases the death of a variable amount of periosteum already detached, torn, and bruised, and so partially devitalised in advance of the infection by the direct violence. Such destruction of any great extent of periosteum seriously interferes with future subperiosteal osteogenesis, and so of course limits the effort at repair of the bone lesion. In cases where in addition bone destruction has been extensive, this would promise, on theoretical grounds at any rate, to prove a serious defect, but practically in flat bones any disability caused thereby is negligible, compared to that of a similar absence of the reparative rôle of the periosteum in compound comminuted fractures of the shafts of the long bones; moreover, it but rarely happens that the periosteum of the two surfaces of the bone are simultaneously destroyed to anything like the same great extent. Again, the failure of subperiosteal formation of new bone means that the size, shape, and walls of the cavity that remains after sequestrectomy will prove much more favourable to its spontaneous healing, and where such appears unlikely or impossible the radical operation necessary

for its obliteration will, on account of this very defect of osteogenesis, be very much the less formidable.

In the majority of cases, however, subperiosteal osteogenesis has to be reckoned with. In long-standing cases the new bone produced reaches an ivory-like hardness and density and a thickness often twice or thrice the thickness of the original layer. Such new bone may be laid down on one—the superficial surface only of the sequestra, and so form the outer wall of the sequestral cavity, or, on the other hand, be formed from the periosteum of both surfaces of the original bone. In the treatment of large cavities it is the former layer only, the outer wall of the cavity, with which the surgeon is concerned. Concurrently with this production of new bone, the nearest surviving original bone at the periphery of the sequestral cavity is undergoing a progressive sclerosis, so that the whole cavity becomes walled in by hard, dense bone.

Where an edge or border of such a bone as the scapula or ilium is struck by a projectile most often merely a piece of it is chipped off, the case following either an aseptic or an infected course; the former usually results in the case of a bullet wound, the latter where the injury is due to a shell fragment. Where, however, the bone edge receives the impact of a bullet that has been travelling in a line parallel with the plane of its surfaces, the full explosive force of the projectile becomes manifest in the frequently terrific comminution and splintering of the bone. The bullet remains lodged among the fragments in some cases; in others it tunnels its way completely through the bone. Such cases often remain aseptic; where, however, they suppurate a very long and narrow tunnel or cavity eventually comes into existence filled with sequestra and walled on either side by bone becoming progressively thicker and of greater density.

C.—Infected Articular Fractures that become Cases of Septic Arthritis as well as Cases of Osteomyelitis

With the arthritic phenomena in their acute stage we are not here concerned; it is rather with the result of the

arthritis—ankylosis, either bony or firm fibrous, that the orthopædic surgeon has to deal. Thus, in addition to one or other of the forms of chronic pathological bone lesions that we have described, we get in these cases obliteration more or less complete of the articular cavity. This ankylosis, whilst adding sensibly to the functional disablement of the limb, very often, by rendering easier the task of obliteration of the osteomyelitic cavity, carries with it at least a partial compensation. It is precisely the cases of chronic disease with excavation of a juxta-articular epiphysis that are the most difficult of all forms of osteomyelitis in which to effect healing; with, however, the elimination of the joint cavity, the bone condition becomes very often as easily cured as if it were located in the middle of a diaphysis.

Many of these cases of infected articular fractures, however, end, not in ankylosis, but in a false joint. In these the chronic stage of osteomyelitis is analogous, both from the pathological and from the therapeutic standpoints, to that described under Type 8 of diaphyseal osteomyelitis associated with non-union.

Infected Cavities of Osteomyelitic Origin; their Varieties and Characteristics

The pathological result common to all the cases and types of chronic traumatic osteomyelitis that we have described is the formation of an infected bone cavity. As regards their size, their shape, their position and the details of their mode of production, these are of infinite variety; as regards their mode of production, however, all may be classified into one of two groups:

(1) Cavities of excavation.
(2) Sequestral cavities proper.

In both classes the original process of cavitation is the work of the projectile. Thus the first stage in the production of either variety is represented by the fracture cavity

that originally exists as the direct and immediate result of the trauma. In all cases this is subsequently modified by the changes, destructive and reparative, that infection and inflammation bring in their train. Practically all cavities—except the smallest—are alike incapable of complete spontaneous healing; in all cases cure is difficult to obtain even by surgical means.

Cavities of Excavation.—These represent the original fracture cavity, enlarged by the destructive carious action of the infection. As we have seen in a few cases, and on a small scale, necrosis may play a part in this process of bone excavation; but, on the whole, such cavities are the result of trauma plus caries. This, then, is the type of infected traumatic cavity of cancellous bone tissue. In dealing with infective osteomyelitis of the short bones and epiphyses, we have seen that the whole bone may be excavated, that various surfaces, articular or free, may become involved by its extension, that the cavity may come to form a complete tunnel, and that most characteristic is its formation when an infected foreign body has been left embedded in a mass of cancellous bone.

The walls of such a cavity are formed of vascular rarefied bone, and lined with infected granulation tissue. Healing by granulation is the only possible course of spontaneous healing; and this, whether because of retention of a foreign body, of slight but progressive activity of the carious process, of premature contraction of the peripheral scar tissue, resulting in inadequacy of blood-supply to the granulations of the central still unhealed part of the cavity, in the majority of cases fails to reach completion.

Sequestral cavities proper are rather more complicated in their mode of formation. The fracture cavity produced by the original trauma undergoes subsequent enlargement in the first place on the completion of sequestration; dead bone that has hitherto formed one wall of the cavity, becomes loosened, and, after complete separation, constitutes a content of the cavity. At the same time the destruction of a layer of living bone by caries—the responsible agent

in sequestration—results in a further slight increase in the size of the cavity. Meanwhile, however, with this increase in dimension in one direction, the limits of the cavity in another are being sharply defined by the appearance of the involucrum.

We have already studied the chief types of sequestral cavities as well as the condition of absence of cavity from failure of subperiosteal osteogenesis. In practically all these different types of sequestral cavity, failure of spontaneous healing, as we have already noted, is almost predestined from the beginning; apart from the question of the presence of foreign bodies and encased sequestra, the incollapsible and sclerotic nature of their walls renders spontaneous healing by secondary adhesion of these walls impossible.

Both varieties of osteomyelitic cavities will undergo a rapid extension of their limits on the occurrence within them of a severe exacerbation of the infection; in cancellous bone the cavity walls will become eroded by acute caries, in sequestral cavities proper the dense sclerotic avascular walls are only too prone to undergo massive necrosis.

The Cloacæ.—The openings through the bony walls of these cavities by means of which they communicate with the track through the soft parts, and hence with the exterior, are called **cloacæ.** In traumatic osteomyelitis there are two varieties of cloacæ, the primary and the secondary. The former represent the original points of entrance and exit of the foreign body in its course through the bone. In the case of cancellous bone these remain as perforations of the outer shell of the bone, which may become slightly enlarged by subsequent caries of their edges, but which practically never develop a width equal to that of the cavity whose mouths they form. In the case of real sequestral cavities the cloacæ exist at first as mere tears or holes in the detached periosteum; with the occurrence of subperiosteal osteogenesis they become better defined, and come to appear as openings through the involucrum. In both cases they persist as natural openings for the drainage of the cavity.

Secondary cloacæ are of later origin; they may arise in several different ways, in cancellous bone by the involvement of a free surface of the bone in an extension of the carious process, in a sequestral cavity by the separation of a piece of necrosed bone that had hitherto formed part of the outer wall of the cavity; these are the two commonest modes of the spontaneous formation of secondary cloacæ. Such may result also, however, from surgical intervention —post-operative or artificially produced secondary cloacæ. In some cases, too, a cloaca is more or less artificially produced by the insertion and retention of a tube or other form of drain through a tear or incision of the original periosteal sheath of the fracture cavity.

Beyond a progressive increase in the thickness of the walls of those that are formed by subperiosteal new bone, these openings in the cavity undergo but little change during the course of the disease.

The Sinuses.—In exactly the same manner sinuses may, as regards the time of their appearance and the mode of their formation, be divided into two classes, primary and secondary.

Primary sinuses represent the partially healed original wounds; secondary, the partially healed tracks that have resulted from an extension of the bone disease with the formation of late abscesses in the soft parts—abscesses that have either burst spontaneously or been incised and drained.

Sinuses causally connected with bone disease vary in length, according to the anatomical site of the cloaca from which they lead. In the case of a subcutaneous bone like the tibia, a sinus can often hardly be held to exist, the external opening of the cloaca itself being directly continuous with the skin opening. On the other hand, in the case of the femoral shaft very often the sinus runs a long and tortuous course from skin to bone, often, too, coming into close relationship with, indeed in many cases actually including in its walls, important nerves and blood-vessels. Where the muscles and skin of a normally well-covered part of a bone, like the shaft of the femur, have been either

cut away by the original trauma or else destroyed by the infection, it becomes to all intents and purposes a subcutaneous bone, and in such again a sinus can very often hardly be said to exist at all.

As a sinus comes in time to consist of a cylinder or tube of dense sclerotic scar tissue lined internally by a layer of granulations, it follows that the natural and inevitable contraction of such tissue results in a tendency, not only to constriction and kinking of the tube at various points in its course, but also to a shortening of its length, an approximation of its outer attachment to the skin towards its inner point of firm fixation to the bone. This leads to a puckering of the skin around the external opening of the sinus, and very often to the formation of a funnel-shaped depression, at the bottom of which, frequently at first sight invisible, because of the irregular nature of this puckering, lies the outer opening or mouth of the sinus. In the case of many sinuses, Nature seems to make an attempt at their healing by the ingrowth of epithelial cells along their granulating walls; indeed, in short sinuses leading into bone cavities it is not uncommon for the whole wall of sinus and cloaca alike to be lined by such epithelial cells.

Most sinuses in time reach a chronic stage beyond which further progress towards completion of healing becomes impossible or at best very slow and uncertain; this partly by reason of the diseased process, incapable of spontaneous change, that exists in the bone, the discharge from which they have to conduct to the surface of the body, in part—and in certain cases where the original *fons et origo mali* has been cured, this is the sole cause of the failure in their healing—because of the dense sclerotic avascular nature of the scar tissue forming their thick rigid walls. Occasionally, however, in cases in which for the time being the infection is latent and the discharge practically ceased, temporary healing of a sinus may take place at any rate over a part of its length by secondary adhesion of its walls approximated by the constricting force of the contraction of their scar tissue elements. In such a case, unless the focus of diseased

or dead bone or the bone cavity possesses another passage for the drainage of its discharge, the accumulation of this discharge will induce an acute exacerbation of the infection.

The Muscles.—Lacerated by the projectile and by fragments of bone, in part destroyed by the infection, incised here and there by the surgeon at various stages, involved in late abscess formation, the muscle surrounding the affected bone in considerable part disappears; what remains is infiltrated with inflammatory products, and penetrated at numerous points by healed and unhealed sinuses. The final result is a sclerosis of such of the muscular tissue as has escaped destruction, and a replacement of the rest by dense masses of scar tissue that directly connect the skin scar with the bone scar. Hence, it will be easily understood that in every case of infected gunshot fracture developing into a case of chronic osteomyelitis, a considerable proportion of the functional disability that remains after final healing is due to this destruction and damage of the muscles surrounding the bone in question.

The Joints.—As we have seen, neighbouring joints may be directly affected in septic osteomyelitis either by direct communication of the infection or by their mere involvement by contiguity in the inflammatory process. The former produces ankylosis, either bony or fibrous, the latter sometimes has a like result, sometimes results in a condition of chronic hydrarthrosis with relaxation and softening of the ligaments—in a more or less flail type of joint.

Limitation of full range of movement of the joint due to extra-articular changes is, however, very common; such results from sclerosis of muscles, of tendons, and even of skin, from fibrotic changes in parts of the joint capsule or ligaments, from mal-union of the original fracture, or from overgrowth by productive osteitis of the diseased articular extremity of the bone, or from any combination of these.

The Skin.—During the long-drawn-out chronic stages of the bone disease, such skin as has been destroyed by the original wounds, or the subsequent infection, is very largely replaced by epithelial proliferation; indeed, as we

have seen, this may even result in an epithelial lining of part of the sinus walls, and even also in short sinuses may commence to pave the bone cavity itself. Very often, however, where the skin loss has been extensive, the process of epithelial repair is brought to a standstill before completion, a typical example of that failure of healing by granulation, the cause of which we noted in Chapter II. (page 30).

The surrounding skin that has survived destruction becomes the seat, however, of certain changes. It becomes infiltrated with inflammatory products, in the earlier stages remains tense, œdematous, and shiny; later, it becomes looser, and, as we have seen, puckered and drawn in around the mouths of the sinuses. The œdema gives way to a general thickening, the result of diffuse infiltration of its subepithelial layers with scar tissue elements. Subjected, too, as it is over long periods to the irritant action of the discharge, it very frequently develops in some cases an acute erythema, in others an acute widespread vesicular eruption, in others, again, a scaly psoriasiform type of chronic eczema.

The Occurrence and Results of Acute Exacerbations of the Infection

Any increase in the activity or virulence of the infection, from whatever cause, will change the whole aspect of the phenomena of chronic osteomyelitis that we have just described as occurring in bone, muscles, joints, and skin. Such relapses to an acute stage are only too common; indeed, as long as the primary cause of failure of healing remains in existence they are to be expected, to be regarded as inevitable. In many cases the provocative cause of such an increase in the intensity of the previously latent infection is obvious; in others not so evident. Among the more obvious causes one of the most frequent is the infliction of a fresh trauma on the infected tissues, *e.g.* the performance of a surgical operation, without adopting precautions to

limit the spread and intensification of the infection, the forcing of a false passage with a probe, a fall resulting in local bruising of the part, or even in a re-fracture of the bone. In other cases the essential factor seems to be connected with some defect of drainage leading to the retention and accumulation of the discharge in the whole or a part of the cavity. This may arise in one of several ways: a sequestrum or foreign body may become impacted in a cloaca or sinus, or blocking of the natural exit may take place more slowly by the gradual occlusion of the sinus, ending in its obliteration over a part of its length, by that process of contraction and adhesion of its walls that we have described; in other cases an inflammatory swelling that is at first absolutely limited to the soft parts may come to result in an intensification of the infective and inflammatory phenomena in the bone by interfering with the passage of the discharge along the sinus.

The existing complicated mass of scar tissue that marks out the limits of the diseased area—the osteosclerotic bone forming the walls of the bone cavity, the dense scar tissue of the sinus walls, the cutaneous scar surrounding the mouths of the sinuses—all form a very ill-nourished and unsuitable ground of defence against an acute infection; as we have seen, such dense unyielding avascular tissue is incapable of becoming the seat of an acute inflammatory reaction, and under the double stress of the inflammation and the infection is only too prone to undergo death *en masse*. Such is indeed what almost invariably happens during an acute attack.

Hence the intensification of the infection and of the inflammation very often results in the occurrence of further necrosis of part at least of the dense walls of the bone cavity as well as of its fibrous contents; as regards an excavation cavity in cancellous bone, however, acute caries and not necrosis is the agent in the extension of its limits. All the phenomena of the original acute attack are thus repeated, the only difference being that, in the meantime, union of the fractured bone has taken place; the case may be regarded

now as one of acute spontaneous osteomyelitis, rather than of traumatic osteomyelitis. However, weakening of the union with re-fracture of the bone from rapid destruction of the bone scar tissue of the union is not so very uncommon. This extension of the bone involvement will lead, as before, to further abscess formation, to the production of new cloacæ and sinuses, and possibly to the occurrence of an acute arthritis ; even to that of pyæmia.

In the soft tissues sloughing and ulceration of the fibrous bands of scar open up fresh tissue to the action of the infection ; acute abscess formation, acute cellulitis, even gas gangrene or tetanus may result.

The skin of the limb again becomes œdematous, tense, red, and shiny ; the cutaneous scar around the sinus exits, unable to resist the combined strain of the infection plus the reaction, rapidly sloughs ; in many cases a sharp attack of erysipelas follows such exposure of the lymphatic spaces of fresh skin to the entry of the infection, whilst acute lymphangitis and lymphadenitis are common occurrences.

Where the patient recovers without the loss of life or limb the resumption of the chronic state on the part of the disease in time produces a condition apparently identical with that obtaining before the attack ; but the bone cavity is now larger and more complicated, the amount of dead bone greater, the sequestra more numerous, one or more extra cloacæ and sinuses have been added, and further damage has been inflicted on muscles and skin, possibly also on joints.

CHAPTER IV

THE EXAMINATION OF CASES OF CHRONIC TRAUMATIC OSTEOMYELITIS

THE greater number of cases of chronic traumatic osteomyelitis that arrive at an orthopædic hospital do so after months, and sometimes after years, of suppuration, continuous or intermittent; many are admitted from civil life; practically all have had one operation, many several operations, performed in the attempt to rid them of their disease, to procure healing of the chronic sinus. In the great proportion of such cases the bone disease has never been properly eradicated, or, where a sufficiently radical operation has been performed, the result has been spoiled by the occurrence of post-operative necrosis, or by faulty after-treatment; in a few the actual bone lesion has been cured, and the cause of the failure of completion of healing is to be found in the soft parts; in still fewer the discharge is due merely to a chronic ulcer, all the tissues being healed except the skin; in very occasional cases the whole wound has completely and soundly healed, and the discharge results merely from a condition of chronic eczema of the skin around the scar.

In all such cases an accurate history, followed by a thorough clinical examination that in turn is supplemented later by a bacteriological investigation of the discharge and a radiographic study of the bone lesion, becomes necessary to put the surgeon in possession of all the essential facts, past and present, of the case, and so to enable him to form an accurate imaginative picture of the actual and whole pathological condition. Such is the first essential of all successful treatment.

The Clinical Examination of Cases of Chronic Osteomyelitis and of Chronic Sinuses

The following is the routine of examination and record adopted by the writer in all such cases:

A.—**Previous History of Fracture.**

(1) Date of wound.
(2) Nature of projectile.
(3) Site and character of fracture.
(4) Presence and character of sepsis.
(5) The nature of the splints and dressings that have been applied.
(6) The nature and date of previous operations.
(7) The size, number, and approximate date of emergence of sequestra.

B.—**Complications.**

(1) The involvement of nerves.
(2) The involvement of vessels.
(3) The involvement of muscles.
(4) The involvement of joints.
(5) The involvement of viscera.

C.—**The State of Union of the Fracture.**

(1) The degree of union, the amount of callus, and of new bone formation.
(2) The presence and amount of shortening.
(3) The presence of angulation or other form of malunion.
(4) The presence of tenderness of the bone.

D.—**The Range of Movement of Joints, Including the Cause of Limitation or Increase of Range.**

E.—**The Examination of Scars and Sinuses.**

(1) Position and general description.
(2) Character and amount of discharge.
(3) The existence of dead bone.
(4) The existence of sequestra.
(5) The existence of a metallic foreign body.
(6) The existence of a cavity.
(7) The existence of a tunnel.

The probe plays a part in the examination that is second only to the skiagraphy, as regards the information it gives, indeed in some cases it results in giving even greater information. But a word of warning is necessary regarding the use of this instrument. In careful hands, even when used with the greatest gentleness, it is capable of bringing about disasters. The writer has seen more than one case of pyæmia result from a rough and careless examination with the probe; also numerous cases of erysipelas, cellulitis, and further extensions of the osteomyelitis. It should be used only where absolutely necessary in eliciting important information, and always most gently. There should never be any attempt to force a passage with it; the false passage produced in probing a sinus can end in results quite as disastrous as a false passage made by passing a sound or catheter through the urethra, and is as much to be avoided.

The clinical types of cases of chronic osteomyelitis vary enormously with the anatomical site of the fracture, the structure of the affected bone, the original extent of the infection, the duration of the disease, the nature and results of previous operation, and many other factors; it is not necessary to attempt to describe all the different types; no two cases are alike; the pathological details of each must be realised as far as possible to the full by clinical and radiographic examination. It is important, moreover, if possible, before operation to relegate each case of chronic sinus to one or other of three great classes:

(1) Cases where there is still dead or infected bone or a bone cavity present.

(2) Cases where the obstacle to healing exists in the soft parts only.

(3) Cases where healing has really taken place, but this is masked to superficial examination by a chronic discharge from the skin.

The first of these classes alone includes actual cases of osteomyelitis; the second and third cases in which, though there has been a bone lesion, this itself is now cured.

It might seem at first sight easy to decide into which of these categories to place any individual case, but frequently this cannot be done with any certainty until during the performance of an operation; many cases of apparently no more than a short sinus in the soft parts, kept from healing by the enormous density and thickness of its walls, will, on later examination or at operation, be found to be associated with a bone cavity or sequestrum. Radiography is of the greatest assistance in the classification of cases from this standpoint. The points on clinical examination alone, however, that will lead one to suspect the persistence of a bone lesion are: tenderness on percussion or palpation of the bone at the seat of fracture, local heat with or without redness elsewhere than around the sinus, complaint of pain in the seat of fracture whilst the degree of union is being tested, a profuse purulent discharge, and the existence of more than one unhealed sinus.

Necrosed bone, when superficial and visible, is diagnosed by its dead white or slightly yellow colour, when deeply placed and out of sight by the characteristic grating sensation it imparts to the probe, and the resonant note percussion which the probe elicits from it; a sequestrum in addition to these furnishes evidence of its mobility; a metallic foreign body gives a higher and more metallic note than dead bone; the presence of a cavity is demonstrated by free movement of the point of the probe from one wall into contact with another; whilst the existence of a tunnel is established either by passing a probe completely

through the bone, or by passing two probes through different sinuses and feeling their points meet between bony walls.

The Radiographic Examination of Cases of Chronic Osteomyelitis

For the most part a study of X-ray plates gives most accurate and detailed information concerning the bone condition ; in some cases of old standing disease, however, with enormously thickened walls, the bone may appear perfectly normal although it contains a large cavity with numerous sequestra, so that radiographic examination should be used to supplement, and not to supplant, clinical examination.

Plates taken in one plane alone are usually deceptive ; the greatest amount of detailed and accurate information will be gathered from a study of two sets of stereoscopic plates taken at right angles to each other with a probe inserted into or through the cavity. By such a method the position and relations of the cavity and various sequestra may be easily determined. A point of great practical importance in the case of tunnels is to make out which is the more slender of the two walls.

In disease of the innominate bone radiography furnishes but a confused picture from which little information is to be gathered.

The Bacteriological Examination in Cases of Chronic Osteomyelitis

Bacteriological examination of the discharge should be a constant preliminary to every operative procedure ; as we shall see, the identity and relative number of the organisms in the discharge should, with the clinical character of the discharge, serve as a guide to the best time to intervene, in order to assure the least amount of post-operative infective reaction.

CHAPTER V

THE CLINICAL COURSE OF CHRONIC TRAUMATIC OSTEOMYELITIS

In this and the succeeding chapters the writer will confine himself to a consideration only of that stage of traumatic osteomyelitis in which, the fracture having united, the infective and inflammatory processes constituting the disease have settled down into chronicity; a chronicity that, indefinite as regards its duration, is inevitably destined to be interrupted at intervals by a series of acute exacerbations of these processes.

If left alone or subjected to merely inadequate surgical treatment, such cases will of themselves progress a certain variable distance towards the accomplishment of healing, but will subsequently reach a point beyond which their pathological processes remain apparently stationary until such time as the onset of the next acute stage. The period between two such acute exacerbations, which varies in length in different cases, is in most a matter of weeks or months, occasionally in old-standing cases a matter of years; almost inevitably, however, as long as bone disease is present, every period of quiescence of the infection sooner or later gives place to and is succeeded by an acute outbreak.

During these long periods of inactivity of the infection there is taking place a slow but progressive series of pathological changes, that clinically become manifested in different ways. Progressive increase in the formation of new bone soon becomes evident clinically, both to sight and touch; in certain places, *e.g.* as in the lower ends of the humerus and femur, such deposit of new bone results in an increasing

limitation of the range of flexion of the neighbouring joint. The acute inflammatory reaction in the bone and soft parts subsiding, other important clinical changes ensue. The seat of union becomes less tender; all redness and œdema gradually disappear; active use, and to a certain extent gradual passive mobilisation, become possible; muscles become softer, joints less stiff and painful; scar tissue hitherto red, dense, and vascular, grows whiter and more supple; except round the mouths of the sinuses the skin becomes less œdematous and less adherent, and again capable of a certain amount of movement on the subcutaneous tissues.

The sinuses themselves become smaller and smaller, some heal altogether, others reach a chronic dilatory stage; with gradual increase in the thickness and density and subsequent contraction of their fibrous walls they draw the skin into numerous puckers and furrows; often the mouth of the sinus, a small red patch of exuberant granulations, lies practically invisible at the bottom of a deep funnel-like inversion of the skin. The sinus itself often becomes lined right down to bone by ingrowth of epithelial cells; other sinuses undergo an alternation of healing and breaking down. The discharge becomes less profuse and less purulent, in time merely a small amount of serous fluid; in this organisms become progressively fewer in number; indeed on examination it will often be found to remain practically sterile over quite long periods.

During this period the general health of the patient is good; he eats and sleeps well, and soon regains much of the strength and colour lost during the acute stage; the temperature remains normal; usually he is able to get about, often to return to work, the sinus causing him no discomfort, but only the annoyance of the daily dressing.

Sooner or later, inevitably comes the change; most often with dramatic suddenness, sometimes the result of a local injury, the rough use of the probe, more usually without any such obvious cause. The actual cause, however, in these attacks is almost always some defect of drainage of the sequestral cavity: a sinus has become an inadequate channel

of drainage for the cavity either because of the impaction in it of a wandering sequestrum or foreign body, or because of its more gradual occlusion by the contraction of the scar tissue within its walls.

Usually, before the appearance of any sign of local inflammation, the temperature suddenly swings upwards; soon, however, the local changes make themselves known. The bone becomes painful and tender; the subcutaneous soft parts swollen, the skin in time intensely red, shiny and œdematous; the edges of thickened skin and of cutaneous scart issue around the mouths of the sinuses, and the walls of the sinuses themselves slough *en masse;* the lymphatic glands draining these tissues become enlarged and tender, but very rarely go on to suppuration; thick green pus makes its reappearance; the patient is acutely ill, gas gangrene, pyæmia, tetanus occasionally supervening. In time an abscess forms, points in one or more places, bursts or is opened, and so lays the foundation of another chronic sinus.

Immediately the inflammatory reaction begins to ebb in its intensity; the temperature comes down; the discharge lessens in amount, and becomes thinner; the sloughs disintegrate and are cast off; the infection gradually sinks to its former level of quiescence and latency. The reparative processes are soon seen to be at work in attempting the repair of the tissue loss and destruction; in appearance there is in time a return almost to the *status quo ante;* in reality, however, there has taken place only too often further necrosis and destruction of bone tissue with a consequent enlargement and complication of the already existing large and complicated sequestral cavity.

Sometimes indeed the recrudescence of the infection arises in and remains limited to the soft parts; an acute cellulitis with abscess formation occurs; in others the skin alone bears the brunt of the intensified activity of the infection, and an acute attack of lymphangitis or more commonly of erysipelas takes place. In cases of epiphysitis or disease of the short bones the neighbouring joint often

comes to be directly involved in the spread of the infective process.

So long as no adequate and radical surgical treatment is undertaken, so long as an encased sequestrum or an unhealed and unobliterated sequestral cavity remains, so long will the life of the patient with chronic osteomyelitis consist of long periods of quiescence of the infection during which the reparative processes are doing their ineffective utmost, periods inevitably and dramatically interrupted at longer or shorter intervals by acute exacerbations of the infection and inflammation that are accompanied by and result in further destruction of tissue.

CHAPTER VI

GENERAL PRINCIPLES OF THE TREATMENT OF CHRONIC TRAUMATIC OSTEOMYELITIS

General Consideration of the Aims and Ideals of Treatment

The ideal at which all treatment of simple fractures should aim may be defined as **early sound union of the bone in normal anatomical position with early recovery of normal function in the limb.** There can be no successful realisation of this ideal without the co-operation of the surgeon and the patient in the fulfilment of two primary conditions :

(1) The replacement and maintenance of the bone fragments in their normal anatomical position.
(2) The early mobilisation, active and passive, of the muscles and joints of the limb.

These two desiderata must be so co-ordinated that the fulfilment of the one does not interfere with that of the other.

In compound fractures, however, the additional element of infection is introduced ; and a third is to be added to the conditions whose fulfilment alone can mean complete success, *viz.* the successful prevention of development of the existing infection, in other words the accomplishment of the prophylaxis of infective osteomyelitis. This book is not concerned with a discussion or an account of the methods whereby such prevention may be effected ; we have already seen in the Introduction that failure on a large scale has occurred, and probably will always occur, in the gunshot fractures of modern war. Such failure, as we have also

seen, entails the most serious consequences—the development of septic traumatic osteomyelitis with all the numerous and serious sequelæ and disabilities of its acute and chronic phases. In addition it adds seriously to the difficulty—very often renders quite insuperable—of successful fulfilment of conditions (1) and (2) alike; it thus renders replacement and immobilisation of the bone fragments difficult or impossible, inflicts serious and often permanent damage on muscles and nerves, and postpones for long periods all passive movement and all active use of neighbouring joints.

Where, however, chronic osteomyelitis is already a *fait accompli*, there should still exist for the surgeon an ideal of treatment towards the realisation of which all his energies and ingenuity should be directed. Such an ideal includes:

(1) The earliest possible final arrest of the infective process, *i.e.* the early attainment of final or permanent healing of the wound.
(2) The promotion of early sound union, a union which, when complete, will provide a sufficiency of strength of bone for the execution of its normal functions of support and leverage.
(3) The earliest possible mobilisation of muscles and joints.
(4) The earliest possible correction of deformities of union.
(5) The earliest possible treatment of complications and residual disabilities.

Now in actual practice the fulfilment of the first of these conditions will in almost all cases precede that of all the others, except the second. Indeed, with this single exception the accomplishment of this first constitutes an almost absolute *sine qua non* of the very feasibility of attempting the fulfilment of any of the others. Hence it follows that in all cases of chronic osteomyelitis the task of pressing, of paramount importance before the surgeon is the attainment the earliest possible moment of a healing of the wound

that will be final; this in the latter stages of all compound fractures becomes indeed a "consummation devoutly to be wished."

The relationship in time that exists between the successful arrest of the chronic septic process and the attainment of sound and functionally useful union varies with the type of fracture. In the majority of cases union takes place, despite the continuance of the infective process—indeed the persistence of its continuous irritation seems to act often as a potent factor in the production of union. In other cases, especially in cases of fracture of the shaft of the humerus, and of one of such a pair of bones as the radius and ulna, in which considerable comminution has been complicated by extensive periosteal destruction, the occurrence of spontaneous union is not to be expected; a pseudarthrosis results, and the attainment of union by surgical means, by the interposition of a bone graft or by other osteoplastic procedure, cannot be ventured upon for many months after the attainment of apparently final healing. In such cases, then, the attainment of union is absolutely dependent on the final elimination of the infective element from bone and soft part.

The Attainment of Final Healing.—In every case the accomplishment of this task, with the promise it bears of freedom from recurrence of bone disease, from the formation of new abscesses, and from the reopening of old wounds, implies and requires in its turn the fulfilment of four conditions:

(1) The elimination, either through their spontaneous emergence or by their surgical removal, of all sequestra and infected foreign bodies.
(2) The surgical removal of all patches of infected carious bone.
(3) The removal of all mechanical obstacles to healing, including not only the obliteration of all cavities or tunnels of bone of even moderate dimensions, but also of all chronic sinuses through soft parts

the complete healing of which is prevented by the excessively dense scar tissue forming their walls.

(4) The prevention of formation, or the removal of all considerable masses, cords, or surfaces of ill-nourished scar tissue—tissue that is only too prone to undergo ulceration and sloughing. This means the reduction to a minimum of all scar tissue connecting skin and bone, and the covering of all surfaces of bone scar with skin of normal structure and thickness in place of the thin cutaneous scar that is so liable to ulcerate.

Although it is easy in writing and in theory to postulate these conditions of complete success in the attainment of a lasting healing, yet in very many cases, especially in cases of chronic disease of the femur, nothing in surgical practice requires a greater expenditure of skill, more careful and experienced judgment, more assiduous attention and constant care over a longer period than the successful accomplishment, by these means, of finality of cure.

The Significance of the Spontaneous Emergence of Sequestra.—We have seen in the chapters on pathology the circumstances and conditions in which alone the spontaneous emergence of sequestra becomes possible. Only, moreover, in the rare cases where all the encased sequestra are of a shape and size to permit of their passage through the cloacæ, and only in cases where there has been no formation of a closed sequestral cavity, so that the sequestra lie free in an open cavity, is their spontaneous appearance at all likely to be followed by permanent, or indeed by any healing. Even in these cases such emergence is always unreliable and haphazard, often delayed, and, when it does occur, often productive of a serious exacerbation of the local infection, of much severe pain and general disturbance, if not of an actual extension of the bone disease.

It is only the surgeon absolutely ignorant of the significance of the pathological condition he is dealing with who will

rely on spontaneity of emergence of sequestra to bring about healing, or who will even wait and see whether a given sequestrum will not come out of itself. As a general rule the emergence of sequestra, whether spontaneous or taking place after various forms of physiotherapy, has no prognostic value whatever. Practically the only exception to this rule is the case of septic amputation stumps, although even here the surgical removal of all sequestra that do not actually of themselves drop off is still absolutely indicated.

The Surgical Removal of Sequestra.—It is indisputable, then, that sequestra should be removed by a surgical operation; the only question that arises is that of the stage at which this operation should be performed.

The sequestra in compound fractures, it should be remembered, are not all of equal age; some date from the time of the injury, others from a later date. Certainly there can be no advantage in delaying the performance of sequestrectomy in cases where the bone has long since united. In such cases one should wait merely to be certain on grounds of thorough clinical and radiographic examination that all dead bone present has separated, and on grounds of clinical and bacteriological examination that the intensity and virulence of the infection has for the moment at least subsided to a satisfactory degree before proceeding with the operation. Of course, in such cases one may be forced to operate for the drainage of an abscess or the removal of an impacted sequestrum before the process of sequestration is complete; but, as a general rule, except in the presence of such urgent signs or symptoms, one should always wait until it is certain that sequestration is complete.

Especially where it seems likely that, in order to effect the removal of all sequestra, much division of involucrum with opening up of new bone to the infection will be necessary, will it be unwise to operate during or immediately following a period of exacerbation of the inflammation, an exacerbation that is often the result of an attempt at spontaneous emergence of a sequestrum. It is in cases, however, at an earlier stage, especially in cases where, because of

extensive periosteal destruction, union seems at a standstill, that difficulties arise. One hesitates to operate for fear of breaking up what union there is; on the other hand, the question presents itself as to whether the local condition is likely to improve without the removal of these dead bone elements. Except in cases where it is obvious that a pseudarthrosis is bound to result, in which the sooner the sequestra are removed the better, it seems wise to wait and watch the case carefully for a few weeks. Where the discharge is draining satisfactorily and there are no signs of extension or exacerbation of the local inflammatory process, where the general condition of the patient is good, and the temperature remains normal, and union is found to be progressing, though but slowly, it seems to the writer better to wait, to allow as much union to take place as possible before intervening. On the other hand, where, apart from the question of union, the case is not doing satisfactorily, the general condition is poor, the temperature high in the evening, and the limb shows signs of further abscess formation, and where there is no appreciable change in the amount of union, operation is indicated; although in some of these cases the mere institution of adequate drainage is all that is needed to lead to the rapid production of union, "very numerous are the cases yet in which, after several months of suppuration, clearing out the infected area of the bone, specially if there have been sequestra, leads to a rapid return of the process of ossification, which previously had come to an almost complete standstill." [1] The occurrence of acute inflammation with or without abscess formation, or of the impaction of a sequestrum, is at all times of course an absolute indication for operation; incision and drainage in the one case, the removal of the sequestrum in the other, becomes necessary.

In cancellous bone sequestrectomy is largely replaced by mere curettage of the infected and diseased bone and granulations—a procedure that is equally necessary, however, in flat bones, and in the shaft of long bones, the pockets and

[1] Broca, *The After-effects of Wounds of the Bones and Joints.*

recesses of the main sequestral cavity of which are almost constantly the seat of an infective caries. The object of thorough curettage is not only or even chiefly the removal of all infected tissue, but also the removal of the surrounding masses of dense scar tissue, that, by interfering with the blood-supply of the granulations, have brought about such a state of stagnation of tissue defence and repair.

The elimination of all dead and diseased bone, then, constitutes the first step in the attainment of healing; in a complicated case—and, as we have seen, many cases in which the shaft of a long bone forms the seat of the disease are very complicated—the accomplishment of this means a long, tedious, and formidable operation. Not rarely, it means a wide opening up of the seat of the disease, and an extensive removal of involucrum—so wide a removal, in certain cases, that it becomes justifiable indeed to proceed at once and resect the little more the removal of which would subsequently become necessary in the performance of the radical operation for the obliteration of the sequestral cavity,—even in a few others to refracture the bone and correct the mal-union. Indeed occasionally in the complete removal of sequestra such a complete osteotomy becomes a matter of necessity and of inevitability rather than of choice; the complete operation was necessary in Case 6, where no real sequestral cavity existed, but the sequestra and infected foreign bodies lay scattered round the displaced ends of the bone, embedded in a huge mass of immature callus. *Vide* Plate VI. (facing p. 56).

The Obliteration of Cavities and Tunnels.—It cannot be stated, however, too emphatically that in all cases where sequestral cavities or tunnels of even moderate size remain, after the removal of dead and diseased bone, healing, alike spontaneous, complete, and stable will not take place. In practically all such the mere removal of sequestra and the prevention of further post-operative necrosis guarantee quite an insufficient assurance of final arrest of the disease. Failure to attempt the obliteration of such cavities and the performance of an insufficiently radical operation for

their obliteration constitute commoner causes of failure of healing than either inadequate sequestrectomy or the occurrence of post-operative necrosis of the freshly opened up bone. In all cases where the sequestral cavity is large it should be understood, and at once taken for granted, that all hope and expectation of finality and stability of healing after sequestrectomy alone, no matter how thorough its performance and how careful the after-treatment, are founded on a misapprehension of the nature of the process of tissue repair.

From our study of the pathology of sequestral cavities it has become obvious that what is required in such cases is a more radical procedure that, after the lapse of an interval, will lead to the obliteration of the cavity or tunnel by the entrance of the periosteum, and the superjacent soft tissues into it, whether this operation be done simultaneously with the sequestrectomy or following it. Any operative procedure that accomplishes this, whether performed in one or two stages, is what we call the radical operation for osteomyelitis. Although this radical operation is a strictly atypical one—indeed the details in each individual case vary with the site, the shape and size, and above all with the relations of the cavity—its aim and principle are single—the substitution of a cavity with at least one soft collapsible wall for a completely rigid-walled cavity, in other words, the attainment of its healing by the process of secondary adhesion of its walls; the general methods of the fulfilment of this aim, however, are two.

METHOD I is applicable especially to cases of cavities of the flat bones and of the shafts of the long bones. It consists in the subperiosteal resection of as much of the wall of the cavity or tunnel as will render possible immediately or in the near future contact and adhesion between the elevated periosteum with its muscular and cutaneous coverings and the bony floor of the remains of the cavity. In many cases of tunnels this resolves itself into a simple wide subperiosteal removal of one wall, either the more slender or the more accessible.

METHOD 2 is especially adapted to cases of cavities of the epiphyses and the tarsal bones, that lie in intimate relation to joints; and also to such other cases in flat bones and diaphyses where the normal covering of soft parts has been destroyed and replaced by scar tissue only. In all such cases a plastic or flap operation becomes necessary; although in the former class of case, if the neighbouring joint be ankylosed, one may be enabled to utilise Method 1.

The performance of this radical operation by one or other of these methods is the only form of treatment or operation that will give uniformly good results. All others that depend for the obliteration of the infected cavity on an attempt at its absolute sterilisation during the operation, followed by packing with various antiseptic pastes and mixtures—whatever the result may be in a primarily aseptic cavity—give results that are not comparable for a moment with those of this radical and much more rational operation. Broca, who has had a large experience of the results of both methods, writes: "In practically all cases (and even, I should add, when used in primarily aseptic cases) late suppuration occurs, and the formation of chronic sinuses with ultimate discharge of the paste. I am almost certain that cases of primary union without the extrusion of the extraneous matter have been quite exceptional, and that, when such extrusion has occurred, the mixture has done no good whatever."[1]

The Occurrence and Prevention of Post-operative Necrosis.—After any operation in an already septic field, a recrudescence of infective activity is to be feared; in these cases of traumatic osteomyelitis this tends to be almost constant in its occurrence, and often of a severity of which present-day surgeons have had no previous experience. Hence in the performance of these operations, either simple sequestrectomy or the more radical procedure we have just described, there is always this ever-present dread—the occurrence of post-operative necrosis, the development of further foci of caries in the bone newly laid open by chisel or

[1] *The After-effects of Wounds of the Bones and Joints.*

curette to the intensified action of the infection. Therefore, in planning the treatment and operations necessary for each particular case, this factor has to be taken into account. Despite the fact that there is no certain means of absolutely preventing such unfortunate sequelæ, yet, where appropriate precautions have been taken, they will be found to be by no means invariable or even necessarily frequent occurrences. In most cases of disease of the femoral shaft in which a new bone surface of great thickness and density has been divided and so exposed to the infection, a certain amount of further necrosis is still unfortunately almost invariable, and this necessitates a further, though usually a very much easier and simpler, sequestrectomy.

In order to obtain a maximum of effectiveness in the prevention of further bone infection, certain precautions must be observed before the performance of the operation, and others taken both during and after its performance.

The time at which the operation is to be undertaken should not be decided on grounds of convenience, either on the part of the surgeon or of the patient; this should be determined by one consideration only, *viz.* the condition of the diseased area. It is only when this has been brought into as nearly aseptic a condition as possible that any operation should be performed—such as simple sequestrectomy, subperiosteal resection of a tunnel-wall, or the plastic type of radical operation—in which new bone tissue is divided and opened up to the infection. The recent occurrence of signs anywhere in or about the wound of acute inflammation or the presence of a profuse purulent discharge should be regarded as absolute contra-indications to the performance of such operations. In addition, however, more active and aggressive steps must be undertaken; wherever anatomical conditions allow, the whole infected area should be subjected for a week or ten days before the operation to a constant or intermittent irrigation with saline or Dakin's fluid, interrupted at intervals for a day at a time for the purpose of bacteriological examination of the discharge. The operation should only be done when, in addition to a satis-

factory appearance on clinical examination, there is a satisfactory bacteriological report—the discharge containing few organisms of any sort, and none actively pathogenic.

The direct application during the operation of antiseptics by irrigation or swabbing has no effect on the subsequent reaction; differences of opinion exist, however, as to the advisability and the results of rubbing Morison's bismuth and iodoform paste (B.I.P.), into the cut surfaces of the bone and muscles. In a series of cases in which this was done, the writer found that, on the whole, the intensity of the post-operative inflammatory reaction was thereby diminished; but the occurrence of several cases in which the paste acted as a foreign body, and delayed healing, led to its abandonment, and the use of the Carrel-Dakin method was found to give better and surer results.

Wherever a freshly divided bone surface is exposed to infection and hence to a risk of necrosis, primary suture of the wound, without or even with drainage, constitutes a direct invitation to the occurrence of post-operative necrosis. It should be made an absolute rule to leave all such wounds widely open. It is a great mistake for any surgeon to suture such a wound in the hope that primary and sound healing will take place, but that if suppuration does occur the sutures can be easily removed, and no great harm will be done. On the contrary, only too often very serious harm in the form of post-operative necrosis results from such an irrational mode of procedure. Secondary closure may be carried out certainly as early as possible, but only when and where the absence of further death and destruction of bone has been demonstrated, or, in cases where necrosis has occurred, only when, after the completion of sequestration, all the dead bone has been removed.

Equally necessary are post-operative precautions, which, when conscientiously carried out, are usually most effective, at the most in preventing, at the least in limiting, the extent of the necrosis. The writer treats the whole open area of bone and muscle surfaces by the Carrel-Dakin method of treating recent wounds; from immediately after

the operation, the whole surface is kept continuously or intermittently moist with hypochlorous acid solution.

This system of pre-operative, operative, and post-operative precautions has been gradually evolved, and found to give far more satisfactory results than any other methods used singly or in combination.

The Causes of Further Delay in Healing after the Performance of the Radical Operation.—Where, however, despite these precautions, bone infection with necrosis has proved inevitable, it is very often confined to superficial layers of those surfaces of the bone from which the periosteum has been stripped. An additional advantage in not closing the wound will now be evident: the surgeon is thus enabled, not only to note the occurrence and the extent of the necrotic process, but to watch the separation of dead bone, and so to prevent premature closing of the wound. Instead of being in the dark as to the extent or even as to the very occurrence of this necrosis, he is in a position, as soon as sequestration is complete, to effect secondary closure with an almost absolute assurance as regards the final elimination of the bone disease.

Besides the occurrence of post-operative necrosis, however, other factors occasionally serve to delay final healing, either before or after secondary closure has been attempted. A patch of cancellous bone that has become infected, and the seat of a progressive caries, is not so easily detected by the eye, although it usually is by the probe. Curettage of this will become necessary.

For other reasons also a slight secondary operation or even more than one may be needed, *e.g.* where the last stages of healing of the wound, despite a successful closure of its deep parts, become stationary, and a chronic ulcer or small sinus surrounded by dense scar tissue persists. Every attempt should be made to get these to heal in order to render more likely a successful aseptic course for their subsequent excision; should, however, their final stage of healing prove too prolonged, excision with primary suture or followed by some simple plastic skin operation will have

to be resorted to, care being taken to perform it under conditions as aseptic as possible.

The Necessity for the Excision of Extensive Scars, especially when Adherent to Bone Scar.—No result in chronic osteomyelitis can be regarded with perfect satisfaction that presents, on the completion of healing, either a large mass or cord of dense scar tissue, connecting skin and bone, or, in the case of a subcutaneous bone like the tibia, a surface of bone scar that is covered over to a considerable extent by a thin adherent cutaneous scar. Sloughing of the fibrous ill-nourished mass in the former case, ulceration of the thin epithelial scar in the latter, are never very far-distant possibilities. Their occurrence sooner or later is almost inevitable; breaking down of wounds in old cases of compound fractures is very commonly due, not to a revival of the infective osteomyelitic process proper, but to the occurrence of such lesions of the soft parts only.

Hence the writer is of opinion that, in chronic osteomyelitis, an ideal result can be claimed only in cases in which, in addition to final elimination of the infective bone element, such defects of healing of the soft parts are conspicuous by their absence, in which no such mass of scar connects skin and bone, and in which all surfaces of bone scar are covered by skin of normal thickness and structure. Before the patient is discharged from hospital, even in certain cases before the mobilisation and use of the limb are permitted, such sources of future trouble should be got rid of. After waiting a few weeks from the completion of healing, one should excise the whole mass of scar tissue; normal muscle and skin should then be drawn together and stitched in layers over the surface of the healed bone. In order to cover a superficial bone surface with normal skin, the excision of the scar will often have to be followed by some simple plastic skin operation.

The Treatment of Residual Disabilities and Deformities after the Attainment of Healing.—After the completion of this stage of final healing, gradual—certainly not forcible—mobilisation and active use of the muscles and joints in the

neighbourhood of the healed bone becomes permissible; but the surgeon is not free to proceed with other operative procedures for some time because of the risk of recrudescence of the infection; any operation on the bone itself should be delayed for from nine months to one year.

CHAPTER VII

DETAILS OF TREATMENT OF CHRONIC TRAUMATIC OSTEOMYELITIS: DETAILS OF CASES

In this chapter the writer proposes to illustrate the practical application of the general principles of treatment outlined in the last chapter to the varying pathological and clinical types of the disease; to discuss at greater length, and in more detail, the methods, operative and non-operative, varied with and modified according to the local conditions and requirements of different cases, whereby alone, in his opinion, the attainment of early sound and final healing of the infective process becomes as nearly assured as at present it is possible to be; at the same time to give details, illustrated by skiagraphs, of a few cases of each type in which cure has been effected by these methods.

It will be readily understood that the ideal result we have in view cannot be attained easily or quickly or at a small price; in most cases it means a series of well-planned and carefully executed operations, each preceded and followed by an interval of assiduous attention and scrupulously carried out precautions. In the great majority of cases two operations will be found necessary, in exceptional cases only one, in a few three or four may prove the only possible minimum. It may seem to be expecting much of the patient to ask him, after the sufferings he has already borne, to submit himself afresh to such a series of further operations, and periods of often wearisome delay between them. But, in default of cure by such rational and systematic means he will assuredly in the long run be called upon to suffer

more : to undergo a series of emergency operations rendered necessary by recurrences of acute activity on the part of the infection ; operations forced on the surgeon by the urgency of the situation, not undertaken by him at a carefully chosen time with a definite curative aim in view ; operations in which the surgeon is not the master of the diseased process, dealing with it as he wishes, but its servant, forced to do what its condition imposes on him ; operations after which the patient is really no further along the road to final cure than before the onset of the acute attack.

Let us recapitulate the steps necessary to the attainment of the ideal at which we are aiming, whatever be the type of case under treatment :

(1) The removal of all sequestra, infected foreign bodies, and infected patches of carious bone.
(2) The obliteration of all rigid cavities and tunnels.
(3) The prevention of post-operative necrosis of bone and of patches of chronic infective caries.
(4) The secondary closure of the wound.
(5) The subsequent excision of any ill-nourished masses of subcutaneous tissue and surfaces of cutaneous scar tissue.

In no case should active treatment be commenced without careful study and thorough realisation of the pathological condition present. Before proceeding to operate the surgeon should acquire a clear mental picture as to the whole diseased area and process, including such exact details as the size, shape, number, and position of the sequestra, the size, shape, and relations of the cavity, the existence of an open or closed cavity, the existence of a tunnel, and, if present, which of its walls on anatomical grounds will prove the more accessible and easy of removal, which on skiagraphic evidence appears the stouter and the more suitable to retain.

Details of the Radical Operation for the Cure of Diaphyseal Osteomyelitis

At a favourable moment, and after the institution of the precautions described in Chapter VI (p. 100), the seat of the bone disease should be explored. As a general rule, it is inadvisable in such cases to use a tourniquet; bleeding vessels should be caught and tied at once. The incision through the skin should be a long and generous one, and run in a direction more or less parallel to the long axis of the bone; short incisions through which the surgeon has to grope in the dark are worse than useless. No really effective and radical operation can be performed by the sense of touch alone or chiefly so; it is absolutely essential for the surgeon actually to see the bone cavity in its entirety and all its details, its numerous hidden pockets and recesses. Such an incision may with advantage be doubled, especially in cases of complete tunnels. Although the excision of all scar tissue in the form of sinuses will require to be carried out before the completion of the operation, yet it is not necessary, in many cases not even advisable, to plan the incision so as to include one or more of these.

The skin incision should be deepened over its entire length through muscle and periosteum, and carried right down to bone. With a sharp-cutting, on no account a blunt-edged, rugine—either Ollier's plane surface rugine or a curved one with a similar sharp edge according to the bone surface to be stripped—the periosteum should now be slowly and carefully elevated from the whole or part of the circumference of the bone at the level of the lesion, and also, for a certain distance, both above and below this level. In stripping the periosteum a midway course should be steered; between, on the one hand, biting too far into the bone with the rugine, and on the other tearing and lacerating the membrane itself, and so interfering with the important part of bone formation, that it has to play in the final restoration of a functionally useful thickness of shaft. In clearing the cloacæ it will be necessary to revert to the use

of the knife; any attempt to do this with the rugine will almost inevitably result in laceration of the periosteum. During this periosteal elevation no bleeding of any account should occur; if a spouting vessel is encountered, this at once shows that the rugine is in the wrong plane; and it is not at all difficult to miss the correct subperiosteal plane, for not only is the periosteum densely adherent to the bone, but the rugine will very readily pass through the fibrous tissue that represents an original tear or gap in the periosteum.

Retractors or bone-levers should now be gently passed round between the bone and the detached periosteo-muscular sheath, and a general survey commenced of the cavity and its openings, and carried out with eye and finger. It is during this and the subsequent stage that a fuller realisation is to be gained of the whole extent and details of the pathological process; it now becomes possible to supplement and correct previous knowledge and conceptions. Still, however, even at this stage, the exact state and extent of the inside of the cavity is as yet a closed book; these can be ascertained only on completion of the next stage of the operation. It is useless, moreover, to yield to the temptation of trying to extract obvious sequestra through the existing cloacæ; not only useless, indeed, but often harmful, as frequently this results in the fracturing off of a small pointed end of spicule of dead bone, which, when thus left caught in a small recess of the cavity, is thereby much more likely to escape observation and removal; such sequestra, in addition, often form the best guide and indication that we have to the existence of such recesses. Where a tunnel or cavity of any size exists, we must next determine which and how much of its walls to remove. In deciding this, not only the relative thickness of the walls, but a consideration as to what portion of the detached periosteum, with its muscular covering, will subsequently be in the most favourable position for entering, filling, and obliterating the cavity must be taken into account; the anatomical relations, and the surgical accessibility of the various walls must also be considered; no hard-and-fast

rules can be laid down even for any special portion of any individual bone.

The surgeon must now proceed to remove the wall decided on, and thus to open up the sequestral cavity. In the majority of cases this is best done with hammer and osteotome or chisel; occasionally a Gigli saw will be found more suitable. During the chiselling, the whole limb should be held steady on a good firm sand-bag by an assistant. In order to avoid complete fracture of the shaft, this becomes all the more necessary during the removal of the last portions of the necessary amount of bone; indeed at this stage it is often wiser to lay aside the hammer and chisel, and use a pair of strong bone-cutting or nibbling forceps. During this removal of bone, by whatever means, gauze strips should be packed between the periosteum and the bone in order to catch any bone-dust that may be produced, and to prevent tiny splinters of bone from getting lost in the tissue. This last is most important; every visible fragment should be carefully removed from the wound before the next is detached with the chisel. Delay in healing after this operation is very frequently due to failure of removal of one or more such bone fragments, which, lying free in the tissues, undergoes necrosis and keeps up the suppuration.

The contents and lining walls of the remains of the cavity must now be carefully inspected, and, in order to carry this out thoroughly, the assistance of a head-light is often indispensable. First, all the obviously free-lying sequestra should be picked out with forceps and removed, care being taken, however, not to break off and leave behind any portions of these that may be engaged in pockets or recesses of the cavity. Where a sequestrum is not yet altogether free, but in part at least locked in such a recess, further removal of bone should be carried out around it, until this recess is well opened up, and the sequestrum becomes quite free. The fibrous and granulation tissue contents of the cavity should next be scraped away with the curette, so as to expose its remaining bony walls. The most meticulously careful examination should now be made by eyes, fingers, and probe

for the existence anywhere in these walls of patches of bone softened by caries, and thus deeply infected—such should be thoroughly scraped, and then laid widely open to form part of the one general cavity; small diverticula and recesses of the main cavity should be similarly treated by further wide removal of their bony walls. A probe should then be passed in through each of the other sinuses and cloacæ in turn so as to identify their inner openings, and make sure that none opens into a recess not yet discovered. Occasionally, too, one will find a small piece of incompletely separated necrosed bone, still forming part of the cavity wall, a small sequestrum or foreign body occupying a cloaca or sinus, or so almost completely surrounded by new bone formation as to be barely visible. All such must be carefully sought out, and when found removed in their entirety.

Apart from such elimination of sequestra and infected bone the further aim of this stage of the operation should be the conversion of the sequestral cavity into a bone surface as nearly plane as circumstances will permit. This demands a wide removal of bone; the groove that results should be left as shallow as possible; no overhanging edge or sharp projecting buttress of bone should be allowed to remain. Such should be the surgeon's first consideration, not the amount of bone that is going to be left; not the sparing of sufficient bone, but the removal of sufficient bone; not even the possibility or likelihood of immediate or subsequent fracture of the remaining thickness of the shaft should deter him. The more thoroughly this is done, the wider and shallower the groove that is made, the more readily will the deep surface of the periosteum come into contact with the bone surface, the surer and quicker will be the process of healing by secondary adhesion of these two surfaces, the earlier the date of final healing of the wound. The occurrence of fracture of the remainder of the thickness of the shaft is no doubt at first sight rather alarming; in point of fact such an accident has but little influence on the prognosis; union in such cases takes place with astonishing rapidity and certainty. Indeed, in many cases of chronic

diaphyseal osteomyelitis accompanied by gross mal-union it is permissible, even advisable, deliberately to fracture through the bone that remains after this stage of the operation is complete, and so to combine the radical operation for osteomyelitis with an osteotomy for mal-union.

The closed cavity in the bone, having thus been converted into a widely open cavity, or rather groove, from the walls of which all necrotic and carious elements have been eliminated, the surgeon should now direct his attention to the soft parts. In turn each of the sinuses should be explored and excised. This is important, inasmuch as they are almost wholly composed of scar tissue which is only too likely to slough during the post-operative inflammatory reaction, and so not only to delay the performance of secondary closure, but, acting as dead tissue, to form a favourable nidus for the growth and multiplication of micro-organisms. Through one of these excised tracks, drainage in what is to be the most dependent part of the limb must now be provided. Care must then be taken to stop all bleeding; hæmostasis as absolute as possible forms a most essential precaution as regards the limitation of the future post-operative intensification of the infection; the occurrence of a hæmatoma or of a reactionary hæmorrhage may quite well be the cause of extensive post-operative necrosis. Except one or two for the retention in position of the drainage and Carrel's tubes, neither deep nor skin sutures should be inserted; into each part and recess of the widely open wound at least one of these latter tubes should be laid, so that no part of the surfaces of the wound, of bone, of muscle, or of periosteum will be able to escape the prolonged or continuous action of the hypochlorous acid. The wound should then be very lightly packed with gauze, and the outer dressing applied and the limb immobilised as for a recent compound fracture; whilst before the patient leaves the theatre the first irrigation through the tubes should be carried out—this largely to prevent their becoming blocked by blood clot.

The After-treatment of the Radical Operation

The immediate after-treatment consists in either intermittent or continuous irrigation of the wound with Dakin's fluid, or with saline solution according to the predilection of the surgeon; this should be carried out most conscientiously. The wound may be dressed at the end of twenty-four or forty-eight hours, the light gauze packing being removed, but care should be taken not to disturb the tubes from their important "strategical" positions.

The reaction after the operation, local and general, varies greatly. With the adoption of the precautions that we have described and especially with the choice of a favourable moment for the intervention, the refusal to take any steps towards the immediate closure of the wound and the successful persistent irrigation of the freshened surfaces, the general average intensity of the reaction has been greatly diminished in the writer's cases. Frequently, and especially in the case of the smaller of the long bones—but also sometimes after extensive radical operations on the tibia and humerus —no visible local reaction, and no rise in temperature have resulted; in other cases the general reaction has been about that of an aseptic operation of corresponding severity; in cases of the femoral shaft a rise in temperature lasting about four days, and then coming down to and remaining normal, has been the rule; in a small percentage of cases the local reaction has been severe and accompanied by a reappearance of thick green pus, and with a certain amount of sloughing of the soft parts and necrosis of bone. The adoption of this technique has, however, decidedly lowered the percentage of cases in which post-operative necrosis has occurred. Unfortunately it has not succeeded in absolutely preventing it in all cases. Cases in which it is to be expected are the following: cases where the local reaction has been severe and accompanied by the formation of the green pus that we have described, cases in which a great thickness of dense compact bone has been divided, cases of extensive operations on the femoral shaft, cases in which an accidental fracture of the

shaft has occurred or in which a deliberate osteotomy of the whole thickness has been performed.

In every case the irrigation should be kept going for at least a fortnight, during which period all tendency to closing of the wound must be discouraged. It has now become of the first importance to detect the existence of necrosis or of progressive caries at any point on the bone surface. Frequent inspections must be carried out, during which an electric torch will be found a great help in the illumination of dark corners and recesses of the wound. By this time the whole surface of the wound, muscle, periosteum and bone will be covered with bright red granulations, the superficial layer of the bone, in part deprived of its blood-supply and exposed to the infection, having undergone by the process of acute caries a rarefaction and conversion into granulation tissue, or, when the infection has been unusually severe in certain places, necrosis. In this latter case the dead white colour of the devitalised bone makes an effective contrast with the red of the granulations, and so such bone is usually easy of detection. Where, however, quite small areas have necrosed these soon become hidden by the exuberant growth of the granulations around them. It should be remembered also that the carious process is in reality merely the necrosis of successive microscopic quantities of bone, and that between such a minute disintegration and the death of larger, more visible portions there must exist intermediate gradations of size, so that the necrosis of very tiny, hardly visible particles must be regarded as almost inevitable in any case. Of course the liberation of such to form correspondingly tiny sequestra will be correspondingly rapid ; but the probability of their occurrence leads to the conclusion that too early closure of the wound before the conversion of this layer of the bone into granulations is well accomplished is inadvisable.

The Secondary Closure of the Wound

At the end of this period of fourteen to sixteen days, and in cases in which it has been possible to keep under observation the whole of the bone surface, and in which no suspicious

patch or spicule has been detected, the surgeon may safely proceed to the closure of the wound. Where necrosis has occurred, however, the completion of the process of sequestration must be waited for; although, if a thin layer of dead bone exists at one spot only, it is often possible to commence the ensuring of secondary adhesion of the granulating bone and periosteal surfaces elsewhere than at this spot, so that partial if not complete closure is usually possible. In all such cases, however, before commencing to close the wound, one should have ample clinical and bacteriological evidence of the relative inactivity and latency of the infection; all signs of acute inflammation should be absent; the discharge slight in amount and serous in nature, and containing but few bacteria, both on microscopic and cultural examination. Where the surface of the bone that has been re-exposed to infection either by being stripped of its periosteum or by its division with the chisel has been so extensive that it is impossible to keep it under observation, then it is best to wait for a longer period before proceeding to secondary closure; in many such cases it is advisable to give an anæsthetic and thoroughly explore, at any rate with the finger and as far as possible with the eye, all such surfaces. In most cases it will be found possible to proceed at once to do at least a partial if not a complete secondary suture of the wound. Such an exploratory procedure, should, however, be preceded by a radiographic examination to determine, if possible, the presence and site of any sequestra or unseparated dead bone that may have resulted after the radical operation. This exploration, moreover, should not be undertaken too soon—in order to give the process of sequestration time to become complete. Nor too late either; for if in any case it be delayed too long, the periosteum commences to deposit a layer or bridge of new bone not against and in continuity with the existing bone surface, but at some distance from it. Where this happens the operation has resulted in the formation of but another tunnel or cavity of smaller dimensions, but certainly one that in most cases none the less will require a second opera-

tion of the same type; so that the obliteration of the original cavity has to be carried out in two stages by two similar operations. This is to be avoided if possible. A similar state of things results in cases where the periosteo-muscular tissue bridging the groove is too tense in relation to the depth of the groove to remain in contact with, and so to adhere to the bone surface or floor—hence the great importance of making the groove or cavity that remains as widely open and shallow as possible by the removal of sufficient bone at the time of the operation.

Secondary closure of the wound can be effected in a variety of ways. We have seen that in some cases it can be successfully accomplished by actual suture of the wound; in the majority of cases, however, it is not necessary to have recourse to this operation; surer results can be obtained by methods of more gradual closure. One by one the Carrel tubes are to be left out; gradually the drainage tube is shortened, and finally omitted altogether. At the same time pressure by means of small splints, pads of gauze, strapping, etc., is to be exerted on the bridge of soft parts, so as to press them gently but firmly into and against the granulating walls of the bone groove. In other cases a similar result may be obtained by means of some extension apparatus that pulls the soft bridge into the desired position and contact with the bone surface. Once complete adhesion has taken place between these two granulating surfaces, the osteomyelitic process may be regarded as cured, the bone itself as healed; further treatment consists in inducing as early and rapid a healing of the soft parts as possible in order to obtain an end result that shows a minimum of scar tissue.

To attain this end similar methods of pressure or extension on the soft tissues should be tried. One such method the writer has found particularly useful in the case of the lower limb is to suspend the leg from a cradle or Thomas's splint, by means of strips of flannel glued on to the skin for two or three inches on either side of the edge of the wound, and so arranged that by pulling on the skin in the direction of the

wound they produce in its neighbourhood a redundancy of skin, and so lead to the approximation of its edges. Where the wound is lateral or posterior, traction in the desired directions can be got by attaching these flannel bands to outrigger metallic bars arranged so as to take the pull of these bands in the appropriate direction, and fixed to the limb above and below the wound by plaster. Where a case is very slow in healing because of the accumulation around it of scar tissue that is becoming increasingly avascular it will often save time to excise the whole sinus and suture the tissues with or without drainage.

The Subsequent Excision of Ill-nourished Masses of Scar Tissue

This is the simplest and easiest part of the whole treatment; it is, of course, not necessary in all cases. It consists merely of the excision some six weeks to two months after the completion of healing—it is wise not to attempt it too soon for fear of a recrudescence of the infection—of the cutaneous scar and that connecting skin and bone. Suture of the wound in layers should then be carried out. Where, however, it is found impossible to bring the skin edges together over the bone a plastic operation will have to be performed; one or two U-shaped flaps of normal skin must be dissected up and sutured in place without tension, or a "caterpillar" graft of skin may be tried, although this necessitates a second stage operation.

The place from which the skin flap has been taken is now to be closed; in some situations where the skin is naturally lax it may be possible to suture this without requiring the transplanted flap to take any of the tension; more often it will have to be covered with Thiersch grafts; or one may trust, by the application of a glue extension apparatus, to obtain a gradual approximation of its edges. Indeed it is a good plan to produce by one of these extension or suspension methods a redundancy of skin in advance arour the scar to be excised so as to render its immediate a direct closure more easy and certain.

The Treatment of the Special Types of Diaphyseal Osteomyelitis

Type I.—**Infective osteomyelitis, following a transverse fracture of the shaft of a long bone, with little or no displacement, little or no comminution.** *Vide* Diagram 6 (p. 54) and Plates I., III., and IV. (facing p. 54).

Cases of this type vary chiefly in the size of their sequestral cavity. This depends on the amount of bone loss, which in these cases is represented by the secondary sequestra. The cavity, which is frequently more of a tunnel, is usually fairly simple and regular, its cloacæ representing the original lacerations of the periosteum by the entrance and exit of the projectile. The treatment of these cases follows closely the lines already laid down in the preceding section.

The following notes illustrate cases of this type:

Case 1.—Chronic osteomyelitis of the shaft of the fibula with an antero-posterior tunnel, containing several small sequestra. Private W. J. W., aged 34, was wounded in the left leg by a piece of shell; a compound fracture of the upper third of the fibular shaft resulted without, however, any displacement.

On examination *December 10th*, 1917.—Patient has had no previous operations; no history of the spontaneous issue of sequestra; bone firmly united; no mal-union; no tenderness of bone; a long scar adherent to the upper half of the fibula, in the centre of which is the mouth of a sinus discharging a little serous fluid; on probing loose dead bone could be felt, and a cavity of some size made out.

X-ray Examination. *See* Plate I. (facing p. 54).

January 29th, 1918.—Radical operation; removal of sequestra and subperiosteal resection of the outer wall of the antero-posterior tunnel; posterior drainage; Carrel-Dakin treatment.

March 10th, 1918.—All wounds healed.

March 18th, 1918.—A tiny spicule of bone has worked its way out.

April 1st, 1918.—Wounds healed. X-ray on this date. Plate II. (facing p. 54).

CASE 2.—Chronic osteomyelitis of the shaft of the fibula ; a considerable antero-posterior tunnel, from which all sequestra had been previously removed without effecting healing. *Vide* Plate III. (facing p. 54).

CASE 3.—Chronic osteomyelitis of the shaft of the humerus with a large sequestral cavity. *Vide* Plate IV. (facing p. 54).

Private M. was wounded by a piece of shell on October 30th, 1915. When first seen on September 8th, 1916, the condition was that shown in Plate IV. ; a large cavity in the upper part of the shaft of the humerus, just below the surgical neck. The patient had had several operations for the removal of sequestra, but the wound had never healed.

September 12th, 1916.—The radical operation performed ; one tiny sequestrum was found in the cavity, which indeed proved to be a side-to-side tunnel, there being a small cloaca on its inner wall. This being a very early case in the writer's series, after the performance of subperiosteal resection of the anterior wall seen in the skiagraph, primary suture was attempted ; the same night the patient's temperature was 105 degrees ; all the stitches cut out and an extensive surface of newly exposed bone underwent necrosis. By October 9th, 1916, this had become a sequestrum, which was removed on that date. In less than a month healing was complete ; the patient was discharged from hospital December 7th, 1916. He has been heard from since, the wound having remained healed.

TYPE II.—**Infective osteomyelitis following a transverse fracture of the shaft of a long bone with little or no comminution but with displacement of the fragments.**

Reference to Diagram 7 (p. 57) and to Plates V. and VI. (facing p. 56) will show that in many cases of this type the sequestral cavity is small, irregular, and subdivided into two separate compartments, each containing secondary sequestra. So small indeed are these compartments in many cases that simple removal of the sequestra, after subperiosteal exploration of the seat of the fracture, alone

suffices to bring about healing; an extensive or any resection of bone is rarely necessary. Case 4, Plate V. (facing p. 56), is an example, however, in which, after sequestrectomy, the spontaneous collapse and healing of the lower of these two compartments have been artificially prevented by the retention within it of a drainage tube; periosteum and bone end have thus been kept apart; subperiosteal deposit of new bone has taken place and resulted in the formation of a permanent tunnel.

Case 4, Plate V.—Chronic osteomyelitis of the femoral shaft with the formation of an antero-posterior tunnel.

Rifleman N. was wounded on June 4th, 1915. A transverse fracture of the middle of the shaft of the femur resulted without comminution, but with overlapping of the fragments. There was a history of alternate healing and breaking down of the wound until September 20th, 1916, when the patient was first seen by the writer. At this time a long sinus was present leading down to the bone; the probe could be felt to pass along a tunnel through the bone; Plate V. represents the skiagraphic picture at this period.

September 29th, 1915.—The radical operation was performed for the obliteration of this tunnel; within three weeks the wound had soundly healed.

Case 5 is a typical example of this variety of diaphyseal osteomyelitis.

Corporal E. S., aged 28, was wounded on October 29th, 1917, by a piece of shell in the left thigh; a compound fracture of the femoral shaft at the junction of its middle and upper thirds resulted. No comminution of the bone took place, merely a transverse fracture with, however, marked overlapping of the fragments. The wound was severely infected, gas gangrene developed necessitating early and extensive excision of muscular tissue in the thigh.

On examination on March 9th, 1918, union was not very firm, although a great thickening of bone around the seat of the fracture was evident; this was very painful on pressure; 1 inch of shortening due to the overlapping was present; in the middle of a long vertical scar, extending

down the back of the thigh, was the mouth of a sinus from which the discharge was serous and slight. The probe entered for 2 inches; a sequestrum, but no cavity, was felt.

March 20th, 1918.—Performance of the radical operation; subperiosteal exploration of the seat of the fracture revealed a small sequestrum lying in contact with each fractured surface, and a certain amount of bony union between the surfaces of the fragments. The sequestra were simply removed, the wound left widely open, and treated by the Carrel-Dakin method. When last seen May 20th, 1918, the cavities had undergone spontaneous obliteration, and the wound had healed except for a superficial ulcer.

Owing to the small size of the subdivisions of the sequestral cavity in this case, no removal of involucrum was necessary to effect their closure.

The next case is an example of this type in which it became necessary to refracture the bone in order to remove the sequestra and infected foreign bodies, so that, in addition to the elimination of the cause of the persistence of the suppuration, a correction of the mal-union was carried out.

CASE 6, PLATE VI. (facing p. 56).—Chronic osteomyelitis of the shaft of the femur, with little comminution, but with great displacement; secondary sequestra and infected pieces of metal embedded in all parts of the callus.

Seven months after the reception of the wound, the amount of union in this case was but slight; a sinus on each side of the thigh remained open, discharging a small amount of thin serous fluid. A probe could be passed from one to the other, but no dead bone could be felt. Enormous antero-posterior thickening existed, and the seat of the fracture was decidedly tender.

The seat of the fracture was then explored and found to be surrounded with a great mass of but slightly calcified callus; there was no sequestral cavity; on reaching the bone ends these were found to be overlapping very considerably; numerous small secondary sequestra and pieces of metal were found surrounding them and embedded in the callus. These were all removed, and in order to bring

the fragment into better position it was found necessary to resect about ½ inch from the lower. The wound was left widely open and drained posteriorly; Carrel-Dakin treatment was instituted at once.

When last seen, about six weeks after the operation, the bone surfaces were completely covered with granulations, no areas of post-operative necrosis had been made out, and union was progressing slowly but satisfactorily.

Here, then, is a case with extreme displacement, but with little comminution—although complicated by the presence of numerous pieces of metal—that well exemplifies the difficulties of operation as well as of judgment that this type may present. No regular sequestral cavity exists in such cases; the attempt at union in the vicious position has resulted in encasing the bone ends, the foreign bodies, and the dead bone in an enormous mass of callus, out of which they literally have to be dug. This in turn involves the breaking up of the union; but, as such union is simply mal-union, this, whilst converting the case into a recent compound fracture, again allows of reposition of the fragments into good alignment; in such case union is likely to be slow, and the occurrence of post-operative necrosis most probable.

TYPE III.—**Chronic osteomyelitis following a transverse fracture with comminution but no displacement.** *Vide* Diagram 8 (p. 59) and Plates VII., VIII., and IX.

These cases are distinguished by the number of their sequestra, primary, and secondary; hence the bone loss is always considerable, the sequestral cavity fairly regular but extensive, usually much more extensive than in cases of Type 1.

The surgical treatment—subperiosteal resection of part of the walls of the cavity with removal of the sequestra follows exactly the lines of the typical operation that we have described in detail.

PLATE VII. CASE 7, represents such a compound fracture with chronic osteomyelitis of the fibular shaft. Plate VII. (facing p. 58) shows the bone united; many sequestra have been previously removed, but healing has not resulted,

owing to the small sequestrum encased in a tunnel that is to be seen in this plate. Removal of this, with subperiosteal resection of the outer wall of the tunnel, was followed by sound healing within three weeks.

PLATE VIII.—The plate forming the frontispiece represents a beautiful example of this type. The large primary sequestrum, surrounded by several smaller secondary sequestra, lies encased within a sequestral cavity, whose walls are of an enormous thickness. The performance of the radical operation with the conversion of so large a cavity into a groove will necessitate the subperiosteal removal of a great quantity of this new bone.

Plate IX. (facing p. 58) represents another case of this type in which, however, the majority of the sequestra have been removed. A large tunnel remains, passing completely through the shaft of the humerus, containing a few small sequestra, and formed by walls of involucrum of great thickness and strength.

TYPE IV.—**Chronic osteomyelitis following a transverse fracture with considerable displacement and considerable comminution.** *Vide* Plate X. (facing p. 58) and Diagram 9 (p. 59).

In cases of this class occur the most complicated of all the sequestral cavities; the sequestra, primary and secondary, are numerous and often large. The involucrum with the lapse of time usually becomes excessively thick, dense, and bulky; union has in most cases resulted from it alone.

The operation for the removal of the sequestra, and the subperiosteal conversion of the sequestral cavity into a wide open groove, is in such cases a formidable one. Not only is a considerable length of shaft involved, but frequently its diameter is greatly increased, the involucral walls of the cavity are very thick and dense, its secondary recesses and pockets are numerous, and contain sequestra that are easily overlooked. In this, more often than in any other type of transverse fracture, a sufficiently radical operation results in a fracture of the shaft; whilst, owing to the thickness and density of the involucrum, secondary post-operative

necrosis is also more common after the radical operation in cases of this type than in any other whatever.

CASE 8.—Plate X. (facing p. 58) shows the end result of a case of this type. When this patient first came under the writer's care chronic osteomyelitis of the femoral shaft had already been present for over one year; Diagram 9 gives a fairly accurate representation of the bone condition at this time: the bone was firmly united, with, however, 1½ inches of shortening, and with marked outward and forward bowing of the femur; a large antero-posterior tunnel existed, filled with large and numerous sequestra.

The radical operation was performed, and so extensive a removal of the enormously thickened involucrum of the outer wall of the tunnel was found necessary, on account of the shape and extent of the sequestral cavity, that the shaft was completely fractured. The limb was straightened and extended; the wound drained behind; Carrel-Dakin treatment commenced. Within two months the bone had firmly united, the amount of new bone formation being surprisingly great. At the end of three months a posterior sinus still existed; this was explored, and a large sequestrum found and removed; it was thus evident that post-operative necrosis had occurred. A month later the sinus was again explored, but was found to be of the soft parts only. It was excised with the scar tissue that led to the bone over which normal muscle and skin were sutured. Thus in five months from the date of the first operation the wound was soundly healed without any excess of scar tissue; all bone disease had been eliminated; the limb had been straightened, and had gained over 1 inch in length, whilst the union was even firmer and stronger than it had been before. This state of the bone is shown in Plate X.

TYPE V.—**Chronic osteomyelitis following an oblique fracture without much displacement of the fragments.** *Vide* Diagram 10 (p. 60) and Plate XI. (facing p. 58).

The amount of the bone loss is slight; the number and size of the sequestra small; a narrow oblique tunnel represents the sequestral cavity.

Frequently in such cases where this tunnel is quite narrow, healing by secondary adhesion of its walls may take place spontaneously after the simple removal of the sequestra. In cases where, however, the tunnel is too wide to admit of this a more radical procedure becomes necessary; the removal of the sequestra, indeed, sometimes requires such a wide opening up of the seat of the disease, that refracture becomes impossible to avoid. Such a combination of circumstances—simple removal of the sequestra plus a resetting of the fracture—frequently results in itself in a successful obliteration of the tunnel with the accomplishment in due time of sound union and of final healing.

Plate XI. (facing p. 58) shows an oblique tunnel filled with sequestra, the removal of which will almost certainly mean a refracture of the bone; subsequent extension and immobilisation of the limb will result in approximation and union of the granulating fracture surfaces, the walls of the previously existing tunnel.

TYPE VI.—**Chronic osteomyelitis, following an oblique fracture with much displacement.** *Vide* Diagram 11 (p. 60).

Such cases, as reference to these diagrams will demonstrate, are characterised by very wide and extensive sequestral cavities or tunnels. Where the amount of necrosis has been slight and the displacement consists almost wholly of an overlapping of the fragments without much separation of their longitudinal axes, a simple removal of the sequestra combined with the subperiosteal removal of the long pointed projecting ends of the two main fragments will frequently be successful in bringing about obliteration of the tunnel and so final healing.

Where, however, overlapping is complicated by a fairly wide separation of these axes, nothing short of a removal of all the sequestra, followed by refracture and reposition of the fractured surfaces in contact, will produce such obliteration and healing.

TYPE VII.—**Chronic osteomyelitis following a fracture with no displacement, but with comminution of the shaft com-**

bined with the destruction of a segment of the periosteal circumference. *Vide* Diagram 12 (p. 62).

The operation here is simple; the original trauma, by destroying the periosteum, has effectually prevented the formation of a closed sequestral cavity, has resulted in an open cavity. The sequestra are easily removed; very often there is no need at all to remove any of the walls of the cavity, and usually the soft parts are easily induced to occupy and fill the cavity.

Where, however, the covering of soft tissues has been destroyed with the periosteum and in part only replaced by scar tissue so that the cavity opens directly on to the surface of the limb, the surgeon will have, after the performance of sequestrectomy, to resort to the plastic type of operation that we will describe later on. *Vide* pages 130, 131.

TYPE VIII.—**Chronic osteomyelitis following a fracture in which extensive, if not total, destruction of a whole segment of the periosteum has taken place along with comminution of the shaft.** *Vide* Diagram 13 (p. 62).

Rarely does a sequestral cavity proper form.

The cases of this type are those that we have already referred to in discussing the proper treatment and time for the removal of sequestra, where union is long delayed. *Vide* page 96.

THE TREATMENT OF CHRONIC OSTEOMYELITIS FOLLOWING INCOMPLETE DIAPHYSEAL FRACTURES

The removal of sequestra and the obliteration of sequestral cavities must in these cases be carried out according to the rules laid down already; the operations are identical in all but their extent with those we have just described for the cases of osteomyelitis following complete diaphyseal fractures.

The following are notes of two illustrative cases.

CASE 9, Plates XII. and XIII. (facing p. 126).—Rifleman L. was wounded in the right leg by a piece of shell, sustaining a compound incomplete fracture of the shaft of the tibia on

October 8th, 1916. A sequestral cavity formed in the lower half of the shaft of the bone, the medullary cavity having evidently been opened into. Numerous pieces of bone had come out spontaneously. On July 7th, 1917, an insufficiently radical removal of the antero-external wall of the cavity had been performed. The patient came under the writer's care on January 1, 1918, when a small sinus was still present; a cavity, but no dead bone, could be made out with the probe.

January 8th, 1918.—This cavity was operated upon, a small sequestrum removed, and the antero-external wall of the cavity widely resected subperiosteally. Carrel-Dakin treatment was commenced.

January 28th, 1918.—No signs of post-operative necrosis; pressure of soft parts into the cavity commenced.

March 23rd, 1918.—The cavity healed, but a chronic superficial ulcer remains; owing to the accumulation of scar tissue around it, its healing has come to a standstill; it requires excision.

CASE 10, Plates XIV., XV., and XVI.—Private V. B. was wounded in the right thigh by a piece of shell on April 9th, 1917; the metal lodged in the cancellous tissue of the lower third of the femoral shaft. This foreign body remained in this position for five weeks before it was removed (*vide* Plate XIV.). A sinus had remained ever since, up to December 1st, 1917, when the patient first came under the writer's care.

December 28th, 1917.—This sinus was explored and found to lead into a bone cavity. The antero-internal wall of this was widely chipped away, the periosteum being carefully spared. Carrel-Dakin treatment was commenced at once. Although an abscess formed in the soft parts owing to inadequacy of drainage, the cavity was healed and a mere superficial ulcer was all that remained by March 1st, 1918. During the next fortnight an attack of acute erysipelas supervened and spread down the leg to the foot; during this period the ulcer finally healed. Scar tissue remains, connecting the skin with the bone; thi requires excision. *Vide* Plate XVI.

PLATE XII. CASE 9.—Before operation. Cavity formation in of the tibia.

PLATE XIII. CASE 9.—After operation. The cavity has been converted into a groove and obliterated by subperiosteal resection of its antero-external wall.

PLATE XIV. CASE 10.—A piece of shrapnel embedded in cancellous tissue of the lower third of the femoral shaft.

PLATE XV. CASE 10.—Shows the cavity that remained after the removal of the foreign body.

PLATE XVI. CASE 10.—After the obliteration of the cavity by subperiosteal removal of its antero-internal wall, healing occurred. Plate taken after healing.

Details of the Radical Operation for the Cure of Osteomyelitis in Short Bones and Epiphyses

We have seen that the chief and characteristic pathological result of infective osteomyelitis in cancellous bone is the formation of "cavities of excavation." Such may or may not contain sequestra or infected foreign bodies. The removal of these, however, is usually comparatively easy; the whole difficulty in these cases consists in the successful obliteration of the cavity. Its spontaneous healing, we have seen, is not to be looked for; as in the sequestral cavity of diaphyseal osteomyelitis, the chief problem before the surgeon is its elimination by some artificial means.

Subperiosteal resection of as much of the walls of these cavities as suffices to convert them into widely opened grooves into which the periosteum and its superjacent soft parts may collapse—a procedure identical with that already described for the obliteration of sequestral cavities in the shafts of long bones—is possible of achievement in two distinct classes of cases.

(1) Where the cavity lies at a considerable distance from the joint cavity, and where there is a sufficiency of tissue to occupy the subsequent groove.

In these cases the treatment is identical in all general respects with that which we have described for sequestral cavities proper. At a like suitable moment, and after adopting identical precautions against a recrudescence of the infection, the cavity is explored, its most superficial wall removed subperiosteally, its remaining walls carefully examined, all dead and diseased bone removed from them with the curette, and the whole cavity converted into a wide groove. The wound is left widely open and the irrigation commenced at once. The after-treatment consists of a period of careful and thorough examination for patches of necrosis or progressive caries, followed by secondary closure of the wound by means identical with those already described in detail.

The following case is illustrative, not only of the method of the cure of such cases, but also of the mischief that results from leaving septic foreign bodies embedded for any length of time in a mass of cancellous bone.

Case 11. Plates XVII., XVIII., XIX., XX.—Chronic osteomyelitis of both the right and left os calcis, with the production in each bone of a cavity the result of the continued presence in the bone of an infected piece of metal.

Private B., aged 22, was wounded in both heels by fragments of a shell on November 10th, 1917. A large piece of metal remained embedded in the left os calcis, a smaller piece in the postero-superior part of the right os calcis (*vide* Plates XVII. and XVIII.). These were *in situ* still on February 2nd, 1918, when the patient came under the care of the writer. There was however a history of the spontaneous emergence of numerous small sequestra from the sinus in each heel. In February and 1918 a sinus discharging thin pus opened on the outer side of the left heel, another on the inner side of the right. In probing the metal in each case he felt embedded in a cavity in each bone.

[illegible] operation—simple removal of the metal from each [illegible] through openings of the wounds. The [illegible] was considerably larger than that [illegible] (*vide* Plates XIX. and XX.). [illegible]

[illegible] slipped very [illegible]

[illegible] the [illegible] superficial [illegible] left [illegible] the [illegible] part of [illegible] as to [illegible] [illegible] [illegible]

PLATE XVII. CASE 10.—A cavity in the left Os Calcis after the removal of a piece of metal.

PLATE XVIII. CASE 10.—A cavity in the right Os Calcis after the removal of a piece of metal.

PLATE XVII. CASE 10.—A cavity in the left Os Calcis after the removal of a piece of metal.

PLATE XX. CASE 10.—Subperiosteal removal of the superior wall of the cavity has resulted in its obliteration and healing; considerable subperiosteal formation of new bone.

March 30th, 1918.—Secondary closure by pressure on the heel flap commenced.

April 12th, 1918.—Both wounds soundly healed. The patient commencing to walk (*vide* Plates XIX. and XX.); both these show a considerable amount of new subperiosteal bone formation.

(2) Where the cavity lies in close proximity to a joint that is, however, ankylosed, the articular cavity having become obliterated.

In such cases the adequate removal of its bony walls is not limited by the presence of an articular danger zone; the case can be treated as if it were a cavity of the middle of a shaft of a long bone; indeed, in many such cases one need not hesitate to divide muscle transversely in order to get the cavity filled, muscle whose only function in connection with the ankylosed joint has been abolished.

Where, however, the cavity lies close to a normal joint, the problem of its obliteration becomes a difficult one. The only alternative to the conservative packing of the cavity daily with gauze, with an occasional scraping in the hope of getting it healed by spontaneous granulation of its walls, is the performance in two stages of a plastic operation. This in some cases is easy of accomplishment; in others it taxes the ingenuity, imagination, and patience of the surgeon to the utmost. The most difficult to get healed are the large cavities that result from the excavation of the epiphyses that lie on either side of the knee-joint—in the lower end of the femur and in the upper end of the tibia.

In all cases of this class the first stage should consist in a thorough curettage of the cavity combined with as wide a sub-periosteal removal of its superficial walls as possible. We are forced in such cases to a compromise, not only by the proximity of the joint, but by the possible failure of future osteogenesis to replace the bone removed. Hence one has to be satisfied with a very much less radical removal of the cavity walls; only too often the cavity still remains, reduced in size, but not converted into a widely open groove,

PLATE XXI. CASE 11.—Antero-posterior view of the upper end of the tibia completely excavated by the process of caries.

PLATE XXII. CASE 11.—Lateral view before operation.

PLATE XXIII. CASE 11.—After operation; obliteration of the cavity by removal of its anterior wall followed by plastic operation.

PLATE XXIV. CASE 11.—Lateral view after operation of the case shown in Plate XXII.

was formed by healthy granulations and the discharge had practically ceased, two musculo-cutaneous flaps were turned in, one from either side, across the face of the cavity, one, the upper with its raw surface facing upwards against the granulations of the overhanging buttress, the other with its corresponding face looking backwards in contact with the lower part of the cavity. These soon adhered, and when last seen, the cavity was obliterated, and the whole wound healed with the exception of two small areas whence the flaps had been taken; these could easily be closed in by Thiersch grafts.

Apart from the question of opening into the joint, one has in these cases to steer a course between, on the one hand, removing too much bone, and so possibly permanently weakening the bone, and on the other removing so little that the flaps have to be cut so long and brought in over so great a distance that they slough or otherwise fail to "take." More experience is required of the reparative powers of the diaphyses after so extensive a removal of one wall as compared with the wonderful powers in this direction possessed by the diaphyses before one can decide how much bone in such cases can be removed with impunity. Thus in this case it will be wise to fit the patient with a walking calliper or a Delbet's walking plaster splint, which he should wear for some time in order to avoid all danger of fracture of the bone.

A similar plastic operation is often necessary to effect the obliteration and healing of open cavities in the shafts of bones the soft tissue covering of which has been destroyed and replaced, though only in part, by scar tissue. Such a condition occurs most commonly in the shaft of the tibia in shell-wound fractures of which the periosteo-cutaneous covering of the internal surface is only too frequently and extensively destroyed. The following case is of this class, and healing was effected by such a plastic two-stage operation.

CASE 13.—Private P. was wounded in the right leg by a fragment of shell and sustained a compound fracture of the tibia and fibula; after many months the fracture united;

an open cavity containing a sequestrum, and with no covering of soft parts at all, was the condition present when he came under the writer's care ; a large area of the skin and periosteum around the cavity had been destroyed, and was only in part replaced by thin epithelial scar, th ingrowth of which over the granulating surface had long since ceased. The sequestrum was removed, the scar tissue completely excised, and the walls of the cavity, which were slightly overhanging, chiselled away, the cavity thus being converted into a wide, shallow, concave surface. Carrel-Dakin treatment was at once commenced and kept up for a fortnight, at the end of which period the groove was carpeted with fresh healthy granulations, no patches of necrosis being detected. Soon after this a large skin flap, left attached at its upper end, was detached from its subcutaneous tissue and shifted laterally, so as to occupy the whole floor of the groove ; a few loose stitches were inserted to keep it in place, but it was subjected thereby to absolutely no tension at all. A small drainage tube was inserted beneath it. No attempt was made to close the gap in the skin left by the transplantation of the flap ; its edges were merely undercut, and then for the next few days subjected to the pull of a glue extension apparatus, such as has been described. Within a fortnight the drainage tube had been gradually shortened and eventually removed, the whole flap had adhered, the cavity was thus completely obliterated, and the bone surface covered by skin of normal structure and thickness, and the edges of the gap, except at one spot, completely approximated and healed. Merely a small unhealed surface, of less than one inch square, remained at the end of five weeks from the date of the first operation.

The Treatment of Chronic Traumatic Osteomyelitis in Flat Bones

Chronic traumatic osteomyelitis in flat bones is to be treated on the general lines laid down for diaphyseal osteoitis. Most cases can be cured by sequestrectomy, by conversion of the sequestral cavity into a wide

open groove into which the periosteum and soft tissues can subsequently be pressed or drawn. In other cases, where the superficial coverings of the cavity have been destroyed, its obliteration will have to be effected by employing an operation of the plastic type.

Hence in all cases of osteomyelitis the sequestral cavity is to be obliterated in one of two ways: in diaphyseal osteomyelitis by adequate subperiosteal resection of its walls, in disease of the epiphyses and short bones by a plastic operation of tissue transplantation; although the former method is applicable to certain cavities in the epiphyses and short bones, whilst in certain cases of open diaphyseal cavities the latter method is alone possible.

In all, no matter where the disease or by what method it is proposed to eliminate it, and thereby effect final healing, the precautions that we have prescribed against the ever-threatening though latent infection must be scrupulously and conscientiously carried out.

According as to which of the two methods of cavity obliteration is employed, one of two general schemes or outlines of treatment that we can draw up applies:

A. Where Subperiosteal Resection of the Cavity Walls is to be employed.

(1) *Period of preparation for the radical operation.*
- (*a*) Incision and adequate drainage of abscesses.
- (*b*) Preliminary removal of sequestra and of foreign bodies.
- (*c*) Preliminary irrigation of the seat of the disease.
- (*d*) Frequent bacteriological examination of the discharge.

(2) *The performance of the radical operation.*
- (*a*) The removal of sequestra and of foreign bodies.
- (*b*) The subperiosteal resection of the cavity walls.
- (*c*) Possibly in some cases the correction of mal-union.
- (*d*) The institution of drainage and the commencement of irrigation of the area of operation.

(3) *The period of after-treatment.*

(*a*) Where no necrosis has occurred :

(1) The mechanical non-operative closure of the wound.

(2) Occasionally the operative closure of the wound.

(*b*) Where necrosis has occurred :

(1) Later sequestrectomy.

(2) Later secondary closure of the wound.

(*c*) Where a persistent small sinus of the soft part remains :

(1) Excision of the sinus followed by suture or simple plastic operation.

(4) *The period of treatment after healing.*

The excision of extensive masses and surfaces of scar tissue followed by suture or simple plastic operation.

B. Where the Radical Operation is to be of the Plastic Type and is to be performed in two Stages.

(1) *The period of preparation for the first stage of the operation.*

(*a*) The incision and adequate drainage of abscesses.

(*b*) The irrigation and cleansing of the wound.

(*c*) The preliminary removal of sequestra and foreign bodies.

(*d*) The frequent bacteriological examination of the discharge.

(2) *The performance of the first stage of the radical operation.*

(*a*) The removal of sequestra and of foreign bodies.

(*b*) The conversion of the cavity into as wide a groove as possible.

(*c*) The commencement of irrigation.

(3) *The period of preparation for the second stage of the radical operation.*

(*a*) The continuation of irrigation.

(*b*) The observation of the granulating bone surface and the detection of the occurrence of necrosis ; where this has occurred, waiting for the separation of the dead bone ; its subsequent removal.

(4) *The performance of the second stage of the operation.*

(*a*) The planning, cutting, and insertion of flaps of the soft tissue, followed or not by

(*b*) The immediate closure of the raw surfaces left after the transplantation of the flaps.

(5) *The period of the after-treatment.*

(*a*) The gradual, mechanical, or non-operative closure of these raw surfaces, or

(*b*) The secondary closure, *e.g.* by skin grafting, of these surfaces.

(6) *The period of treatment after healing is complete.*

It is rarely either necessary or feasible to perform excision of scar tissue after this type of radical operation.

A certain number of cases both of diaphyseal and of epiphyseal osteomyelitis present on the completion of healing at the site of the previous disease an amount and thickness of bone tissue insufficient to perform unaided the normal function either of support or of leverage of the bone. In the great majority of these cases this defect is the result of primary destruction of bone and of periosteum ; in some few cases it is conceivable that it may arise directly from the excessive removal of bone at the radical operation.

Whether in such cases a sufficiency of new bone will be laid down eventually, we are not as yet in a position to know with any certainty ; until such happens, however, precautions against the occurrence of fracture of the bone at its weak spot should be taken.

INDEX

Abscess, acute, formation following exacerbation of osteomyelitis, 81
separation of periosteum from bone by, following infected gunshot fracture, 67
Albee, rôle of bone-graft transplants, 14
Ankylosis, following septic arthritis, 78
resulting from arthritis following upon gunshot fracture of joints, 72, 73
Antiseptic pastes, packing bone-cavities with, 99
Areolar tissue, 4, 12
Arthritis, following infected gunshot fractures, 68
septic, and later ankylosis, following infected gunshot fractures of joints, 72, 73
followed by ankylosis, 78
Aseptic irritants, caries due to, 17

Bacteria surviving in recesses of war wounds, 29
Bacteriological examination of cases of chronic osteomyelitis, 82, 83, 100
Bismuth and iodoform paste, application during operation for chronic osteomyelitis, 101
Blood supply, poor, in scar-tissue, its results of, 48
Bone, acute ulceration or caries of, following infected gunshot fracture in
cancellous, acute caries of, in exacerbations of osteomyelitis, 80
cavities of, excavation following infective osteomyelitis of, 72
closed in, 73, 74
curettage of infected and diseased bone and granulations in, 96, 97
Bone, cancellous, diseased, curettage of, 102
inflammation of, caries in, 17, 18
septic osteomyelitis of, necrosis resulting from caries in, 17
structure, 5
Bone-cavities, classification, etiological and bacteriological, 25
development, following infected fractures of short bones and epiphyses, 65
development in chronic osteomyelitis, 69
formation, 24
infected in osteomyelitis, elimination by radical operation, 46
formations of scar-tissue in, 46
obliteration in chronic traumatic osteomyelitis, 97, 98
methods, 98, 99
of excavation, 73, 74
extension of, 75
following osteomyelitis of cancellous bone, obliteration, 127; difficulties in, 129
method of healing, 74
walls of, 74
of fractures, aseptic, lining wall, 35
packing with antiseptic pastes, 99
persistence after gunshot fractures, 55, 56
retention of infected missiles in, 65
with rigid walls, failure of healing in, 30, 31, 45
Bone-cavity, accumulation of discharge in, setting up acute exacerbation of osteomyelitis, 80
closed, in osteomyelitis, failure of healing of, 45

Bone-cavity, open, in osteomyelitis, failure of healing of, 44
walls of, necrosis of in acute exacerbation of osteomyelitis, 80
removal in operation for diaphyseal osteomyelitis, 108
Bone
cancellous, 5
septic osteomyelitis of, 17
cells, 1
compact, Haversian canals of, 4
more frequent occurrence of bacterial necrosis in, 12
immediately underneath periosteum, structure, 4, 5
sequestra of, bone-earth content, 22
structure, 4
destruction of, following infected gunshot fracture, 67
following infected gunshot fractures, terminal events in, 68, 69
development and growth of, 5
diseased, destruction and damage of muscles surrounding, 78
earth content of sequestra of compact bone, 22
earths, 2
fragments, detached in simple fractures, viability of, 12, 13
necrosis, following gunshot fractures of flat bones, 71
replacement and sound union of, after simple fractures, 91
graft transplants, necrosis, 13, 14
viability, 12, 13
growth in length of, 6
infected, removal in operating for diaphyseal osteomyelitis, 110
inflammatory disease of, how differing from that of other tissues, 8, 9, 10
loss, failure of replacement of, 42
in oblique fractures of shaft, 60, 61
in transverse fractures of shaft, 56, 57, 58
matrix, 2
necrosed, and sequestra, changes taking place in, 21, 22
diagnosis, 85
separation of, 18
new, formation, after gunshot fractures of flat bones, 72
in case of gunshot fractures, 50
Bone physiology, 1
scar and scar-tissue, difference between, 32, 33
scar, formation of, 32
reconstruction and remodelling in complete diaphyseal fractures, 40
young vascular, 36
conversion into dense osteosclerotic, 36
splinters and fragments, retention of projectiles among, in gunshot fractures, 70
tissue, and soft tissue, repair in, resemblances between, 33
at seat of fracture, insufficiency of, 42, 43
caries of, 9, 10
compact, association of rarefaction with reparative effort in, 33
cultivation *in vitro*, 13
first production, subperiosteal, 6
further destruction following acute exacerbations of osteomyelitis, 89
incompleteness of natural repair in osteomyelitis, 42
inflamed, necrosis of, 9, 10, 11
proper, 1, 2
chemical constitution, 2
rarefaction, resulting from subacute caries, 16
repair of, 31
aseptic, modes of, 34, 35
and in presence of infection, 33
failure due to presence of infected foreign body or sequestrum, 44
failure of spontaneous completion in presence of infection, 44
in infective osteomyelitis, modes of, 41, 42
in presence of infection, in infective osteomyelitis, 40
total destruction by acute caries, 14
Bones, flat, characteristics of compared with those of long and short bones, 69
effect of impact of various projectiles on, 70
infected gunshot fractures of, 69
structure, 5
general structure, 1

Bones, human, chemical composition of ash in, 2
long, complete gunshot fractures of shafts of, 49
incomplete fractures of shafts of, 63, 64
shafts or diaphyses of, 5
short, and epiphyses, gunshot fractures of, results, 65, 66, 67
and epiphyses, osteomyelitis in, radical operation for described, 127
infected fractures of, 64, 65
structure, 5
Bony union following gunshot fractures, 55
promotion of, in chronic traumatic osteomyelitis, 92, 93
Broca, bone-graft transplants, 13
packing bone-cavities with antiseptic pastes, 99
Brodie's cold abscess of tibia, 18

Callus, 36
development following fractures, 55
external, formation in fractures with displacement, 36, 37, 39
Canaliculi, 1
Caries, acute, of cancellous bone, in exacerbations of osteomyelitis, 80
resulting in total destruction of bone, 14
and necrosis, contrasts and connection between, 17
due to aseptic irritants, 17
situations under which appearing, 17, 18
in osteomyelitis, 14
of bone following gunshot fractures, 66
of bone-tissue, 9, 10
necrosis resulting from, 17
process of, sequestration a beneficial result of, 19
results of, 18, 24
subacute, resulting in rarefaction of bone-tissue, 16
Carrel's tubes, application to wound for drainage after radical operation for diaphyseal osteomyelitis, 111
Carrel-Dakin method, application after operating for chronic osteomyelitis, 101, 102
treatment in chronic osteomeylitis, 132
Cartilage bones, 5
Cellulitis, acute, following exacerbations of osteomyelitis, 81
following acute exacerbations of osteomyelitis, 89
Chemicals, necrosis of bone-tissue from action of, 11
Chemiotaxis, positive, in relation to tissue-repair, 26, 27
Cloacæ, 59, 60, 75
development of, 55
in cancellous bone, 75, 76
in sequestral cavities, 75
primary, 75
secondary, 76
varieties of, in traumatic osteomyelitis, 75

Dakin's fluid, application to infected area in chronic osteomyelitis, 100
irrigation with, after radical operation for diaphyseal osteomyelitis, 112, 113
Delbet's walking plaster splint, 131
Diaphyses of long bones, 5
reparative powers of, 131
Dobrowolskaja, cultivation of bone-tissues *in vitro*, 13

Eczema, chronic, scaly psoriasiform type developing in skin in neighbourhood of chronic osteomyelitis, 79
Empyema, chronic, failure of healing of cavity with rigid walls in, 31, 45
Epiphyses, 5
and short bones, gunshot fractures of, results, 65, 66, 67
osteomyelitis in, radical operation for, described, 127
gunshot fractures of, frequent in the war, 65
infected, 64, 65
ossification of, 6
Epithelial cells, in relation to tissue-repair, 26
Erysipelas following acute exacerbations of osteomyelitis, 81, 89
Erythema, acute, development, in skin in neighbourhood of chronic osteomyelitis, 79
Erythroblasts, nucleated, 3

False joints resulting from infected articular fractures, 73

Femur, chronic osteomyelitis of shaft of, with formation of antero-posterior tunnel, treatment, 119
with little comminution but great displacement, treatment, 120, 121
complete excavation of lower end of, 130
shaft of, sinus from, 76
Fibroblasts in new granulation tissue, 27
Fracture-cavities with displacement and multiplicity of fragments, alteration of relation of zones in, 37–39
cavity, communication with wound of soft tissues, 55
in simple fracture, three zones of, 35, 36
three zones of, 54
Fracture, insufficiency of bone-tissue at seat of, 42, 43
oblique, of shaft, without comminution or displacement, sequestral cavity of, 60
of bone at weak spot, prevention after operations for osteomyelitis in flat bones, 135
simple, non-union of, 42
union of, mechanical obstacles to, 42
transverse, infected without comminution or displacement, sequestral cavity of, 54
of shaft, with comminution and displacement, sequestral cavity, 58, 59
with comminution, but without displacement, 58, 59
with little or no comminution, little or no displacement, 53, 54
without comminution, but with displacement, 56, 57
sequestral cavity of, 56, 57
Fractures, aseptic, bone-cavities of, lining wall, 35
compound, uninfected, kind of projectiles causing, 48
pathology of, 47, 48
detachment of osteoblasts in, 50
diaphyseal, reconstruction and remodelling of bone scar in, 40
Fractures, incomplete, of one half of circumference of shaft, with oblique or comminuted fracture of other half, 63
of shafts, medullary canal opened to infection, 64
of long bones, 63, 64
medullary canal unopened to infection, 64
infected, of shaft, destruction of periosteum in, 61
of short bones and epiphyses, 64, 65
latent, infection at seat of, from retention of projectiles, 48
oblique, of shaft, size of sequestral cavities occurring in, 61
without comminution, but with displacement, sequestral cavity of, 60, 61
simple and compound, agency of periosteum in union of, 52
mode of repair in, 35
non-union in, pathological types, 43
viability of detached bone-fragments in, 12, 13
with displacements, formation of external callus in, 36, 37

Glue extension apparatus, applica- to limb after radical operation for diaphyseal osteomyelitis, 115, 116
Granulation tissue, conversion into scar tissue, 27, 28
new, fibroblasts in, 27
in acute inflammation, 27
repair by, 29
Gunshot fracture, diaphyseal, osteomyelitis following, cases treated by radical operation, 117–125
oblique, with much displacement, treatment of chronic osteomyelitis following, 124
without much displacement of fragments, treatment of chronic osteomyelitis following, 123, 124
previous history of in cases of chronic traumatic osteomyelitis, 83
state of union in cases of chronic traumatic osteomyelitis, 83

Gunshot fracture, transverse, with comminution, but no displacement, treatment of chronic osteomyelitis following, 121
with considerable displacement and considerable comminution, treatment of chronic osteomyelitis following, 122, 123
with destruction of segment of periosteum with comminution of shaft, chronic osteomyelitis following, 125
fractures, compound, prevention of development of infection in, 91
uninfected, classes of, 48, 49
infected, acute ulceration and caries of bones following, 66
developing into cases of osteomyelitis, 49–63
method of bony union in, 50, 55
of flat bones, 69
pseudarthrosis following, 93, 96
of epiphyses and tarsal bones, frequent in war, 65
of flat bones, comminuted, 72
spontaneous healing following, 70
of joints, followed by septic arthritis and, later, ankylosis, 72, 73
terminating in false joint, 73
persistence of bone-cavities after, 55, 56
simple, treatment, ideal to be aimed at in, 91
transverse, of shaft of long bones, treatment of infective osteomyelitis following, 117, 118
wound of soft tissues, communication with fracture cavity and sequestral cavity, 55

Hæmorrhage, prevention, in stripping periosteum, 108
Haversian canals, 4
system, 40

Inflammation, acute, appearance of granulation tissue in, 27
Involucrum, cylinder of, bony union in gunshot fracture formed by, 51,
origin, 51, 52

Jaw, phosphorus necrosis of, 11
Joint surfaces, proximity of osteomyelitic focus to, 65
Joints, affection of, in septic osteomyelitis, 78
destructive changes in, following infected gunshot fractures of epiphyses, 67, 68
gunshot fractures of, sequelæ resulting from, 72, 73
limitation of full range of, due to extra-articular changes, 78

Lacunæ, 1
Lamellæ, circumferential, 5
Lymphangitis, acute, following exacerbations of osteomyelitis, 81
following acute exacerbations of osteomyelitis, 81, 89

Macewen, Sir W., areolar tissue, 4
bone-graft transplants, 13
formation of external callus, 37
origin of osteoblasts, 7
osteogenetic powers of periosteum, 50
Machine-gun bullets, effect of impact on flat bones, 70
Marrow, 3
cells, 3
tissue, 3
Medullary canal, opened and unopened to infection, in incomplete fractures of soft bones, 64
spaces, 6
Membrane bones, 5
Missiles, infected, retention in bone-cavities, 65
Muscles and joints, early mobilisation, after simple fractures, 91
surrounding diseased bone, destruction and damage of, 78
Myelocytes, 3
Myeloplaxes, 3

Necrosis and caries, contrasts and connection between, 17
following infected gunshot fractures of short bones, 66
of bone fragments in gunshot fractures of flat bones, 71

Necrosis of bone-tissue, 9, 10, 11
aseptic, 12, 13
bacterial, 11, 12
causes, 11
from action of chemicals, 11
of walls of bone cavity in acute exacerbations of osteomyelitis, 80
post-operative, appearance of green pus in cases of, 112
occurrence and prevention of, 99, 100, 101
risk of, 101, 102
resulting from caries, 17
slight, sequestral cavities with, 57, 58

Ollier's plane surface rugine, 107
Operations badly performed, setting up acute exacerbation of osteomyelitis, 79, 80
Ossein, 2
Ossification, direct or intramembranous, 6
endochrondral, 6
Osteitis, post-typhoidal, 18
Osteoblastic granulation tissue, formation after acute stage of infection in gunshot fractures, 55
Osteoblasts, accumulation beneath periosteum, 33
activity of, result, 32
adhesion to detached periosteum in fractures, 52
detachment in fractures, 50
nutrition of, conditions interfering with, 42
origin of, 7
in zone of fracture cavity, 36
proliferation of, 16
vegetative capacity of, 13, 14
Osteoclasts, origin of, 15
Osteogenesis, sub-periosteal, 19
Osteosclerosis, 9, 10, 32
Osteotomy, accompanying radical operation for diaphyseal osteomyelitis, 110
necessity for in certain cases of chronic traumatic osteomyelitis, 97
Osteomyelitis, acute and chronic, resulting from gunshot fractures of flat bones, 70
exacerbations of, 79
after chronic course, 87, 88
causes, 79, 80
Osteomyelitis, acute exacerbations of complications and sequelæ, 80, 81, 89
caries in, 14
chronic, development of bone-cavities in, 69
forms most difficult of cure, 73
closed bone cavity in, causes of failure of healing in, 45, 46
destructive phenomena of, 10
diaphyseal, radical operation for accompanied by osteotomy, 110, 111
radical operation for, cases described, 117–125
described, 107–111
secondary closure of wound after, 113–115
subsequent excision of ill-nourished masses of scar-tissue after, 116
resulting in non-union, 73
special types of, treatment, 117–125
results of X-ray examination in, 117–124
focus of, proximity to joint surfaces, 65
incompleteness of natural repair of bone tissue in, 42
infected gunshot fractures developing into cases of, 49–63
infective, repair of bone-tissue in presence of, 40
open bone cavity in, failure of healing, 44
reparative phenomena of, 26
septic, affection of joints in, 78
of cancellous bone, necrosis resulting from caries in, 17
spontaneous, sites for development of, 64
traumatic, chronic, attainment of final healing in, 93
cases of, bacteriological examination, 82, 86, 100
clinical types, 84, 85
X-ray examination, 82, 85, 86
clinical course, 87–90
examination of cases, 82
clinical method adopted, 83
final arrest of infective process in, 92
hopeless outlook in untreated cases, 90
in short bones and epiphyses, radical operation for, described, 127

Tarsal bones, gunshot fractures of, frequent in the war, 65
Thomas's splint, suspension of leg in, after operation for diaphyseal osteomyelitis, 115
Tibia, Brodie's cold abscess of, 18
cancellous tissue of upper epiphysis of, converted into cavity, 130; method of obliteration of cavity, 130, 131
Tissue-repair by first intention, 28
granulation, 29
failure of, 30
factors in, 26, 27
incompleteness of, causes, 30
irritant factor in, 26
phenomena of, 26–28
reaction, following infection by gunshot fractures, 66
Trauma, freshly infected, cause of acute exacerbation of osteomyelitis, 79, 80
Tuberculous disease of bone, necrosis resulting from, caries in, 17

Vesicular eruption of skin in neighbourhood of chronic osteomyelitis, 79

War (the), frequency of gunshot fractures of epiphyses and tarsal bones in, 65
War wounds, bacteria surviving in recesses of, 29
Wounds, aseptic, process of repair in, 28
clean-cut aseptic, healing of, 26
primary suture after operating for chronic osteomyelitis, risk of, 101
suppurating, method of healing in, 29

X-ray examination in cases of diaphyseal osteomyelitis, 117–124
in chronic traumatic osteomyelitis, 82, 85, 86

LONDON: H. K. LEWIS & CO. LTD., 136 GOWER STREET, W.C.1
Printed by Hazell, Watson & Viney, Ld., London and Aylesbury.

Scar-tissue, ill-nourished masses of, excision after radical operation for diaphyseal osteomyelitis, 116
ill results of poor blood supply to, 28
Scars, examination of, in cases of chronic traumatic osteomyelitis, 84
Secondary adhesion, healing by, 29,—failure of, 30
Sequestra and necrosed bone, changes taking place in, 21, 22
aseptic, 20, 23
encased, 23, 24
spontaneous emergence, 23
treatment, 109
formation after comminuted gunshot fractures of flat bones, 72
following infected gunshot fractures, 55
free-lying, removal, 109
infected, 20, 23
of compact bone, bone-earth content, 22
primary, early and later, 20
removal of, when contra-indicated, 108
secondary, 21
formation, following infected gunshot fractures of short bones, 66
serrated borders of, 21
spontaneous emergence in chronic traumatic osteomyelitis, significance, 94
tertiary, 21
ultimate fate of, 23
varieties of, in traumatic osteomyelitis, 20, 21
Sequestral cavities, 73, 74, 75
cloacæ in, 75
extension of, 75
failure of spontaneous healing, 75
nature of, 74
obliteration, 130, 131, 133
of greatest size, type of fracture in which found, 61
opening up, 109
persistence of, untoward results from, 56
with slight necrosis, spontaneous obliteration of, 57, 58
cavity, communication with wound of soft tissues, 55
Sequestral cavity, conversion into plane bone surface, 110
of infected transverse fracture without comminution or displacement, 54
of oblique fracture of shaft, without comminution but with displacement, 60, 61
without comminution or displacement, 59, 60
of tranverse fracture of shaft, with comminution and displacement, 58, 59
with comminution but without displacement, 58, 59
without comminution but with displacement, 56, 57
without comminution or displacement, 54
Sequestration, phenomena of, 18, 19
process of, duration in different kinds of bone, compared, 19, 20
result of caries, 18, 19
Sequestrectomy, time for performance of, 95, 96
Sharpey, perforating fibres of, 5
Shell or bomb fragments, cases of fracture due to, followed by suppuration, 48
Shrapnel balls, effect of impact on flat bones, 70
Sinus from shaft of femur, 76
Sinuses, condition of, during stage of chronic osteomyelitis, 88
examination of, in chronic traumatic osteomyelitis, 84
healing of, 77
primary, 76
secondary, 76
Skin, changes in, following chronic infective osteomyelitis, 78, 79
incision in before radical operation for diaphyseal osteomyelitis, 107
Soft tissues and bone-tissue, repair in, resemblances between, 33
pressure or extension on, after radical operation for diaphyseal osteomyelitis, 115
Sub-periosteal layer of bone, blood-supply of, 7, 12
cause of death of, 12
operation, osteogenesis of periosteum after, 53
Suppuration following in cases of fracture from shell or bomb fragments, 48

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

NOV 8 22 NOV 20 1933
FEB 23 '23
1928
MAY -1 1934
SEP 16 '24
JUN 13 1935
NOV -6 '24
NOV 27 1943
JAN 18 1927
JAN 31 1928
DEC 10 28
APR 22 '29
SEP 9 29
MAR 17 '30
OCT 28 1931
OCT 29 1931
JAN 30 1932
NOV -6 1933

M684 White, J.R. 50316
W58 Chronic traumatic osteomyeliti
1919

NAME	DATE DUE
D. Wolfram	11-15-43

50316